This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
L341 .H227 1861 STOR
A treatise on the diseases of the nervou
24503407329

LANE
MEDICAL L
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
SEMPER VIRENS
1885
Gift
Dr. [illegible] McClanahan

my dear friend S. WEIR
phia, not only because he
laborer of twenty years'
acquirements as a physician,
ist, are such as to place him
ose who work in the most
of medical science.

125

A TREATISE

ON THE

DISEASES OF THE NERVOUS SYSTEM.

BY

WILLIAM A. HAMMOND, M. D.,

SURGEON-GENERAL U. S. ARMY (RETIRED LIST); PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM IN THE MEDICAL DEPARTMENT OF THE UNIVERSITY OF THE CITY OF NEW YORK; MEMBER OF THE AMERICAN NEUROLOGICAL ASSOCIATION AND OF THE NEW YORK NEUROLOGICAL SOCIETY; OF THE NEW YORK COUNTY MEDICAL SOCIETY; OF THE NEW YORK MEDICO-LEGAL SOCIETY; OF THE AMERICAN PHILOSOPHICAL SOCIETY (PHILADELPHIA); OF THE ACADEMY OF THE NATURAL SCIENCES (PHILADELPHIA); FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA; FELLOW OF THE AMERICAN ACADEMY OF ARTS AND SCIENCES (BOSTON); CORRESPONDING MEMBER OF THE ANTHROPOLOGICAL INSTITUTE OF GREAT BRITAIN AND IRELAND; HONORARY MEMBER OF THE ROYAL MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH; OF THE BRITISH MEDICAL ASSOCIATION, ETC., ETC.

WITH ONE HUNDRED AND TWELVE ILLUSTRATIONS.

SEVENTH EDITION, REWRITTEN, ENLARGED, AND IMPROVED.

"Est quoddam prodire tenus, si non datur ultra."—HORACE.

NEW YORK:
D. APPLETON AND COMPANY,
1, 3, AND 5 BOND STREET.
1881.

S
LANE LIBRARY

Entered, according to Act of Congress, in the year 1876,
By D. APPLETON & CO.,
In the Office of the Librarian of Congress, at Washington.

Entered, according to Act of Congress, in the year 1881,
By D. APPLETON & CO.,
In the Office of the Librarian of Congress, at Washington.

227
381

I dedicate this volume to my dear friend S. WEIR MITCHELL, M. D., of Philadelphia, not only because he is my friend and fellow-laborer of twenty years' standing, but because his acquirements as a physician, and especially a neurologist, are such as to place him among the first of those who work in the most important department of medical science.

725

PREFACE.

THE first edition of this work was published in 1871, and in the preface I then wrote:

"In the following work I have endeavored to present a Treatise on Diseases of the Nervous System, which, without being superficial, would be concise and explicit, and which, while making no claim to being exhaustive, would nevertheless be sufficiently complete for the instruction and guidance of those who might be disposed to seek information from its pages. How far I have been successful will soon be determined by the judgment of those more competent than myself to form an unbiased opinion.

"One feature I may, however, with justice claim for this work, and that is, that it rests to a great extent on my own observation and experience, and is therefore no mere compilation. The reader will readily perceive that I have views of my own on every disease considered, and that I have not hesitated to express them."

The work remained without essential change till the sixth edition, published in 1876. It was then entirely remodeled and greatly enlarged.

This, the seventh edition, has also received extensive additions.

Among the chief of these is a considerable amplification of th chapter on cerebral congestion, the introduction of a chapter o myxœdema, of others on syphilis of the brain, the spinal cord, an the nerves; on the symptomatology of cerebral and cerebellar lesions, and a new section on diseases of the sympathetic nervous system. Material additions have also been made to the chapters o locomotor ataxia, progressive facial atrophy, chorea, epilepsy, neuralgia, and to many others to less extent. I have also added t the chapters on the symptomatology of cerebral and cerebella lesions a translation of the Diagnostic Points given by Nothnage in his remarkable work on the "Topical Diagnosis of Brain Diseases."

For much of the new matter I am indebted to Dr. Labadie Lagrave, the accomplished editor and translator of the French edition of this work, published in Paris in 1879. The chapters o nervous syphilis, on the symptomatology of cerebral lesions, and o the pathology of the sympathetic nerve, are, except as regards th matter included within brackets, literal translations of his article on these subjects.

I have omitted from this edition the chapters on insanity, an this mainly for the reason that I am now engaged in the preparation of a special treatise on that important subject, in which th physiology and pathology of the mind will receive much fuller consideration than could be given in a book combining the whol subject of neurological medicine. Notwithstanding this omissio the present edition contains about forty more pages than the last.

An Italian translation, under the editorial supervision of Professo Diodato Borrelli, Professor of Pathology and Clinical Medicine i the Royal University at Naples, is now going through the press.

It would be affectation to say that the evidences which have been given, both at home and abroad, of the appreciation of my work, have not been a source of gratification. It will be my effort to show in the future that the favor awarded to my endeavors to bring the subject of neurological medicine before the profession and students is not disregarded.

43 West Fifty-fourth Street, New York,
May 1, 1881.

LANE
MEDICAL LIBRARY
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
1885
Gift
Dr.F.C.McClanahan

Dr. A. M. GARDNER.

SECTION V.

DISEASES OF THE SYMPATHETIC NERVOUS SYSTEM.

CHAP. PAG

I.—PATHOLOGY OF THE CERVICAL SYMPATHETIC, ... 86

II.—NEUROSES OF THE CERVICAL SYMPATHETIC, ... 86

Migraine, or Hemicrania.

III.—PATHOLOGY OF THE THORACIC SYMPATHETIC, ... 87

IV.—PATHOLOGY OF THE ABDOMINAL SYMPATHETIC, ... 87

Symmetrical Gangrene of the Extremities.

SECTION VI.

TOXIC DISEASES OF THE NERVOUS SYSTEM.

I.—PLUMBISM, ... 88

II.—ALCOHOLISM, ... 89

III.—BROMISM, ... 91

IV.—HYDRARGISM, ... 91

V.—ARSENICISM, ... 92

LIST OF ILLUSTRATIONS.

FIG.			PAGE
1.	Cephalohæmometer,	*Hammond*,	20
2.	Æsthesiometer,	“	21
3.	Lombard's Differential Calorimeter,	“	24
4.	Lombard's Thermo-Electric Pile,	“	25
5.	Dynamometer,	*Mathieu*,	26
6.	Dynamograph,	“	27
7.	Duchenne's Trocar,	*Hammond*,	28
8.	Stöhrer's Battery,	“	29
9.	Three Cells *in situ*,	“	30
10	Miliary Aneurism of Brain,	*Bouchard*,	107
11.	“ “ “	*Hammond*,	107
12.	Atheromatous Artery of Brain,	“	109
13.	Diagram explanatory of Paralysis in Cases of Cerebral Hæmorrhage,	“	113
14.	Diagram explanatory of Crossed Paralysis,	“	115
15.	Cerebral Arterial Thrombosis,	*Heubner*,	140
16.	Cerebral Capillary Embolism,	*Virchow*,	158
17.	“ “ “	“	158
18.	Cerebral Convolutions	*Broca*,	201
19.	Agraphia,	*Hammond*,	214
20.	Dynamographic Tracing of Patient affected with Multiple Cerebral Sclerosis,	“	296
21.	Dynamographic Tracing of Patient affected with Multiple Cerebral Sclerosis,	“	297
22.	Malignant Tumor of Brain,	*Otis*,	312
23.	Aneurysmal Tumor of Brain,	*Prof. W. R. Smith*,	319
24.	Hand of Patient with Athetosis,	*Hammond*,	328
25.	“ “ after Photograph from Dr. Hubbard,		331
26.	Hand of Patient with Myxœdema,	*Hammond*,	339
27.	Portrait of Patient with Myxœdema,	“	340

FIG. PAGE
28. Motor Cerebral Centres in Man, ... *Labadie-Lagrave*, . 353
29. Cerebral Lesion, ... *Landouzy, Labadie-Lagrave*, . 356
30. Cerebral Lesion, ... *Landouzy, Labadie-Lagrave*, . . 355
31. Internal Capsule, External Face, . . . *Labadie-Lagrave*, . 362
32. Internal Capsule, Internal Face, . . . “ “ . 363
33. Diagram of, explaining Phenomena of Lateral Hemiopia and Crossed Amblyopia, . . . *Charcot and Labadie-Lagrave*, . . 370
34. Patient with Probable Atrophy and Sclerosis of Cerebellum, ... *Hammond*, . 379
35. Morbid Anatomy in Cervical Pachymeningitis, . *Joffroy*, . . 444
36. Deformity caused by Cervical Pachymeningitis, . *Charcot*, . 445
37. Deformity caused by Chronic Spinal Meningitis, *Hammond*, . . 452
38. Spinal Cord in Infantile Spinal Paralysis, . . *Charcot*, . 473
39. “ “ “ “ “ “ . . “ . . 473
40. “ “ “ “ “ “ . . “ . . 474
41. Altered Nerve-Cells of Cord in Infantile Spinal Paralysis, ... “ . . 476
42. Muscle in Infantile Spinal Paralysis, . . *Hammond*, . 477
43. “ “ “ “ “ . . . . “ . . 477
44. “ “ “ “ “ . . . . “ . . 477
45. “ “ “ “ “ . . . . “ . . 478
46. “ “ “ “ “ . . . . “ . . 478
47. “ “ “ “ “ . . . . “ . . 483
48. “ “ “ “ “ . . . . “ . . 483
49. Atrophy of Muscles in Spinal Paralysis of Adults, “ . . 493
50. “ “ “ “ “ “ “ “ . . . 503
51. Patient with Pseudo-Hypertrophic Spinal Paralysis, “ . . 507
52. “ “ “ “ “ . “ . . 507
53. “ “ “ “ “ “ . . 510
54. “ “ “ “ “ . “ . . 510
55. Glosso-Labio-Laryngeal Paralysis, ... “ . . 520
56. Writing of Patient affected with Glosso-Labio-Laryngeal Paralysis, ... “ . . 522
57. Glosso-Labio-Laryngeal Paralysis, . . “ . . 523
58. “ “ “ “ . . . . . . “ . . 524
59. Progressive Muscular Atrophy, ... “ . . 535
60. “ “ “ . . . . . *Duchenne*, . 537
61. “ “ “ . . . . . *Friedreich*, . . 538
62. “ “ “ . . . . . *Duchenne*, . 540
63. Spinal Cord in Glosso-Labio-Laryngeal Paralysis, *Charcot*, . . 552
64. Progressive Facial Atrophy, . . . *Hammond*, . 561

FIG. PAGE
65. Progressive Facial Atrophy, ... *Lande*, . . 563
66. " " " . . . . . . " . . 563
67. " " " . . . . . . " . . 563
68. Muscular Fibre in Progressive Facial Atrophy (Longitudinal Section—normal), ... *Hammond*, . 567
69. Muscular Fibre in Progressive Facial Atrophy (Longitudinal Section—abnormal), ... " . . 567
70. Muscular Tissue in Progressive Facial Atrophy (Transverse Section—normal), ... " . . 567
71. Muscular Tissue in Progressive Facial Atrophy (Transverse Section—abnormal), ... " . . 567
72. Diagram explanatory of Anæsthesia in Lesions of Cord, ... " . . 572
73. Section of Spinal Cord in Sclerosis of Lateral Columns, ... *Charcot*, . . 591
74. Section of Spinal Cord in Lateral Sclerosis, . " . . 591
75. " " " " " " " . . " . . 591
76. Section of Medulla Oblongata, ... " . . 591
77. Deformity in Amyotrophic Lateral Spinal Sclerosis, " . . 594
78. Section through Medulla Oblongata in Amyotrophic Lateral Spinal Sclerosis, ... " . . 599
79. Writing of Patient with Locomotor Ataxia . *Hammond*, . . 608
80. Dynamographic Tracing of Patient with Locomotor Ataxia, ... " . . 609
81. Dynamographic Tracing of Patient with Locomotor Ataxia, ... " . . 609
82. Superior Extremity of Healthy Humerus, . *Charcot*, . . 621
83. " " " Diseased Humerus of Patient with Locomotor Ataxia, ... " . . 621
84. Transverse Section of Spinal Cord, . . . *Lockhart Clarke*, . 625
85. Longitudinal Section of Spinal Cord, . . " " 626
86. Section of Spinal Cord in Locomotor Ataxia, . *Pierret*, . . 632
87. Sclerosis of Columns of Goll, ... " . . 637
88. " " " " " . . . . . . " . . 637
89. " " " " " . . . . . " . . 637
90. " " " " " . . . . . . " . . 637
91. Tumor of the Spinal Cord, ... *Charcot*, . 656
92. " " " " " . . . . . . " . . 656
93. " " " " " . . . . . . *Leyden*, . 659
94. Cortical Substance of Brain in Hydrophobia, . *Hammond*, . . 678
95. Nuclei of Pneumogastric and Hypoglossal Nerves in Hydrophobia, ... " . . 679
96. Root of Pneumogastric Nerve in Hydrophobia, . " . . 680
97. Neuroglia-Cells of Cord in Hydrophobia, . " . . 680

FIG.			PAGE
98.	Contractions in Hysteria,	*Charcot,*	757
99.	" " "	"	765
100.	Catalepsy (after Photograph from Dr. Early),	*Hammond,*	770
101.	" " " " " "	"	770
102.	Induced Catalepsy,	"	774
103.	Ecstasy,	*Bourneville,*	781
104.	Hystero-Epilepsy,	*Hammond,*	787
105.	" "	"	788
106.	" "	*Charcot,*	790
107.	" "	"	791
108.	" "	*Bourneville,*	792
109.	Writing of Patient with Multiple Cerebro-Spinal Sclerosis,	*Hammond,*	802
110.	Writing of Patient with Anapeiratic Paralysis,	"	809
111.	Exophthalmic Goitre (after Photograph from Dr. J. B. Crawford),		815
112.	Diagram showing the Anatomical Divisions of the Spinal Cord,		663

DISEASES OF THE NERVOUS SYSTEM.

INTRODUCTION.

THE INSTRUMENTS AND APPARATUS EMPLOYED IN THE DIAGNOSIS AND TREATMENT OF DISEASES OF THE NERVOUS SYSTEM.

DISEASES of the nervous system, like those of the heart, lungs, and larynx, require special means of investigation and treatment. In no department of medical science has progress been more decided during the last decade than in that class of affections considered in this treatise, and undoubtedly a great deal of the advancement is due to the instruments and apparatus by which scientific research in this direction has become practicable.

In the present chapter I propose to describe the instruments and apparatus employed in the diagnosis and treatment of diseases of the nervous system, and to explain the methods by which they are used.

THE OPHTHALMOSCOPE.

The ophthalmoscope consists essentially of a concave mirror perforated in the centre, and of a double-convex lens. Several modifications of this arrangement are in use, but the simplest instrument is, in my opinion, the best for ordinary use, and this is Liebreich's, though, when very great exactness is required, as, for instance, in determining the depth of an atrophic excavation of the optic disk, Dr. Loring's ophthalmoscope is far preferable to any other.

Liebreich's ophthalmoscope consists of a polished steel mirror about one and three-quarters inch in diameter, concave, and perforated in the centre by a hole about the one-twelfth of an inch in diameter. The edges of this aperture are beveled, so as to afford as little obstacle as possible to the passage of the rays of light to the eye of the observer.

The mirror is set into a bronze ring with a handle, and there is attached also to this ring a clip for holding a concave ocular lens, which in some conditions of refraction, either in the eye of the patie or that of the observer, is necessary in order to produce the requis divergence of the parallel rays emanating from the patient's eye, a thus render the image of the fundus distinct. A direct image is th obtained. The lamp, which should furnish a steady flame, is placed the side of the patient's head corresponding to the eye to be examine and the eye of the observer very close to that of the patient. Thi process gives a very satisfactory view of the fundus with the optic disk and retinal vessels, but requires care, and is more difficult than that b which the inverted image is obtained.

In this case the observer illuminates the fundus with the ophthal moscopic mirror, and then interposes between the mirror and the eye double-convex lens which he holds lightly between the thumb an finger, resting the ring-finger on the forehead of the patient, so as t make the hand steady, the little finger being disengaged so as t be employed in raising the eyelid if necessary.

The object-lens should have a focal distance of about two inche and it should be held so as to bring the focus on the pupil. The lam is placed behind and a little to one side of the eye to be examined. I order to see the optic disk, the patient is told to look at the ear of th observer on the side opposite to the eye being examined. In this wa the axis of vision is directed inward, and the optic disk readily brough into view.

These examinations are made in a room lighted only by the lam used in the processes. It is sometimes necessary to dilate the pupi with atropia, in order to obtain a view of the disk, but experience an tact will generally enable the observer to dispense with this rather dis agreeable procedure.

Ophthalmoscopic examinations require the observer to possess very thorough acquaintance with the anatomy of the eye, and also wit the science of optics. Unless these qualifications are enjoyed, it will b much better to send the patient to a competent ophthalmic surgeon fo an examination than to rush to hasty conclusions based on the mos thorough ignorance. The real value of ophthalmoscopy in diseases o the nervous system is in danger of being disregarded through the scio lism of pert pretenders, who read papers and write memoirs without eve having seen the optic disk to recognize it.

Bouchut[1] gives the following list of abnormal conditions which ar of importance in the diagnosis of diseases of the nervous system :

Papillary congestion ; peri-papillary congestion ; papillary anæmia partial or general ; phlebo-retinal flexuosities ; venous pulsation in th retinal veins ; dilatations of the retinal veins ; retinal varices ; phlebo retinal hæmostases ; phlebo-retinal thromboses ; phlebo-retinal aneu rism ; hæmorrhages into the retina and choroid. The diseases in which he thinks ophthalmoscopy is valuable as a diagnostic means are—the several varieties of cerebral meningitis ; cerebral hæmorrhage ; chronic encephalitis ; cerebral softening ; meningeal hæmorrhage ; chronic hydrocephalus ; tumors of the brain ; contusion, commotion, and compression of the brain ; general paralysis ; atrophy of the brain ; chronic myelitis ; locomotor ataxia ; tetanus ; epilepsy ; essential convulsions ; insanity, and several others of less importance.

---

[1] "Du diagnostic des maladies du système nerveux, par l'ophthalmoscopie," Pari 1866, p. 15.

To these may be added cerebral congestion, general and partial ; cerebral anæmia ; and the various forms of sclerosis affecting the brain and spinal cord.

CEPHALOHÆMOMETER.

Although this instrument is intended for experiments on the lower animals, it enables us to arrive at very definite conclusions relative to the condition of the cerebral circulation. I first described it in a paper read before the New York Medical Journal Association in 1868, and shortly afterward published in the *New York Medical Gazette.*[1] It was devised in somewhat different form, independently of each other, by Dr. S. Weir Mitchell and myself, his being made first in point of time. The instrument consists of a brass, or iron, tube nickel-plated, which is received into a round hole made in the skull with a trephine. Both ends of this tube are open, but into the upper end is secured another brass or iron tube, the lower opening of which is closed by a piece of very thin sheet India-rubber, and the upper opening by a brass cap, into which is fastened a glass tube. This inner arrangement contains colored water. To this glass tube a scale is affixed.

This second tube is screwed into the first till the thin India-rubber presses upon the dura mater, and the level of the colored water stands at 0, which is in the middle of the scale. Now, when the quantity of blood in the brain is increased, the liquid rises in the tube, being pressed upward by the elevation of the thin rubber closing the lower opening ; when the quantity of blood is lessened, the liquid falls by its own gravity.

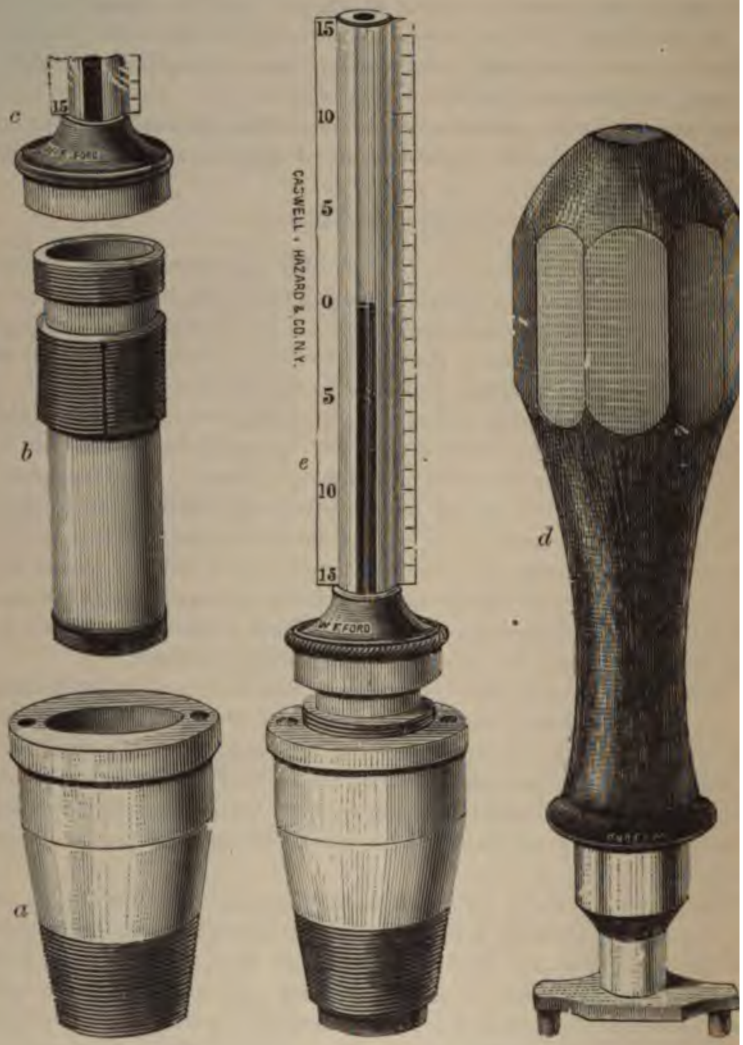
The various parts of this instrument, as made by Mr. Ford, of this city, are shown in Fig. 1 (*a*, the first tube, which is to be screwed into the opening in the skull ; *b*, the second tube, closed at the lower end with thin sheet-rubber and containing the colored water ; *c*, the cap to which is cemented the glass tube ; *d*, the wrench for securing the first tube into the opening in the skull ; *e*, the instrument with all its parts in their places).

It was by this instrument that I was enabled to demonstrate, in the most conclusive manner, that during sleep the amount of blood circulating in the cerebral vessels is much less than during wakefulness, to show the effect of bromide of potassium in lessening the amoun blood in the brain, of the sulphate of quinine in increasing it, etc.[1]

---

[1] Also *Journal of Psychological Medicine*, January, 1869, p. 47.

Fig. 1.



ÆSTHESIOMETER.

The æsthesiometer is an instrument for the purpose of determin the degree of tactile sensibility possessed by the patient. It was vised in 1858 by Dr. Sieveking,[1] of London. Its value in cases of aberrations of sensibility depends upon the fact, ascertained by Dr. E. H. Weber, that the capability of distinguishing two impressions, made upon the skin simultaneously, varies in different regions of the body according to the distance they are apart. In sensitive regions, as the end of the finger, the two points of a pair of dividers can be distinguished at about the twelfth of an inch apart, while in the middle of the back only one point is felt, though they are two inches apart. In accordance with this principle, the æsthesiometer is used to determine the sensibility of the skin in various diseases, it being well known that this is subject to variation.

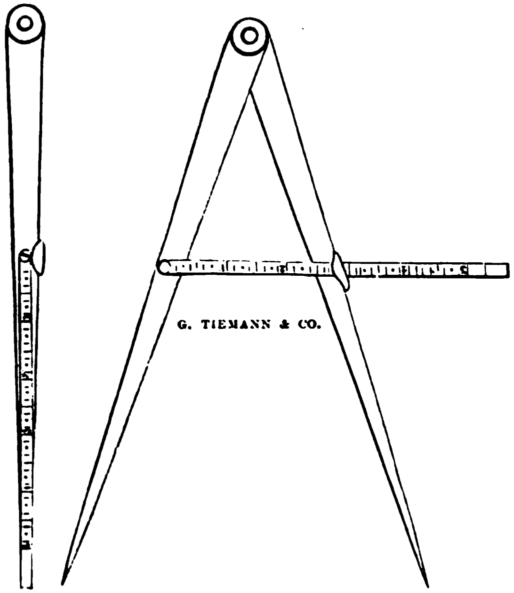
---

[1] "Sleep and its Derangements," Philadelphia, 1869, p. 317.

Thus, when the sensibility is intact, two points, touching the back of the hand at the same time, can be distinguished as two points when separated an inch. If, in examining a patient, we should find that, when the two points were two inches apart, the patient felt but a single impression, we should know that he had lost sensibility in the cutaneous nerves of that part of the body.

Dr. Sieveking's æsthesiometer is nothing more than a beam-compass. It consists of a rod of bell-metal four inches in length, graduated into inches and tenths of an inch. At one end is a fixed steel point; another steel point is made to slide upon the beam, and can be fixed at

FIG. 2.



---

[1] *British and Foreign Medico-Chirurgical Review*, January, 1858, p. 281.

any distance from the first by a screw which works at the top of t slide.

In 1861[1] I described an æsthesiometer which I believe was the fi used in this country. It consisted of a pair of dividers, to one arm which the arc of a circle, in brass, was affixed. This arc was divided as to measure tenths of an inch. A short time since, I suggested Mr. Stohlman, the instrument-maker, a modification of this instrume which for convenience is, I think, superior to all others. This, closed, for the pocket-case, and open, as in use, is seen in the acco nying woodcut (Fig. 2),[2] and need not be further described.

The minimum normal distances at which the two points of t æsthesiometer can be distinguished in different regions of the body a stated in the following table:[3]

Point of the tongue	½	a line.
Palmar surface of the third finger	1	"
Red surface of the lips	2	lines.
Palmar surface of second finger	2	"
Dorsal surface of third finger	3	"
Tip of the nose	3	"
The palm over the heads of the metacarpal bones	3	"
Dorsum of tongue, one inch from the tip	4	"
Part of the lips covered by the skin	4	"
Border of the tongue, an inch from the tip	4	"
Metacarpal bone of the thumb	4	"
Extremity of the great-toe	5	"
Dorsal surface of the second finger	5	"
Palm of the hand	5	"
Skin of the cheek	5	"
External surface of the eyelids	5	"
Mucous membrane of the hard palate	6	"
Skin over the anterior surface of the zygoma	7	"
Plantar surface of the metatarsal surface of great-toe	7	"
Dorsal surface of the first finger	7	"
On the dorsum of the hand over the heads of the metacarpal bones	8	"
Mucous membrane of the gums	9	"
Skin over the posterior part of the zygoma	10	"
Lower part of the forehead	10	"
Lower part of the occiput	12	"
Back of the hand	14	"
Neck under the lower jaw	15	"
Vertex	15	"

---

[1] "A Clinical Lecture on Chronic Myelitis," delivered in the Baltimore Infirma March 16, 1861, *American Medical Times*, June 15, 1861, p. 379.

[2] First described by me in the *Journal of Psychological Medicine*, October, 1868, 830.

[3] This table is quoted from Müller's "Physiology," translated by Baly, London, 184 p. 752.

Skin over the patella	16 lines.
" " sacrum	18 "
" " acromion	18 "
The leg, near the knee and foot	18 "
Dorsum of the foot, near the toes	18 "
The skin over the sternum	20 "
" " five upper vertebræ	24 "
" " spine near the occiput	24 "
" in the lumbar region	24 "
" " middle of the neck	30 "
" over the middle of the back	30 "
The middle of the arm	30 "
" " thigh	30 "

THERMOMETER.

The thermometer is of use for the purpose of determining variations of temperature in different parts of the body. It should be graduated in tenths of a degree, and be held upon the part subjected to examination, so long as the mercury continues to rise or fall. Comparative determinations must be made under precisely similar conditions.

BECQUEREL'S DISKS.

By means of these little instruments very slight variations of temperature can be ascertained. They consist of an extremely thin plate of copper about the size of a half-dime, soldered to a thin rod of bismuth. This latter is contained in a small tube of hard rubber furnished with a handle. The disks are two in number, and by means of delicate silk-covered wires are in communication with the poles of a galvanometer. If a lower extremity, for instance, is subjected to examination, one of the disks is placed upon it and the other upon the corresponding part of the other limb. If the temperature of both limbs be the same, the needle of the galvanometer remains quiet; if either be warmer than the other, the needle is deflected to the north or south according as one or the other limb has the higher temperature. By this apparatus very much less than the hundredth of a degree of temperature can be determined with absolute accuracy.[1]

DR. LOMBARD'S THERMO-ELECTRIC DIFFERENTIAL CALORIMETER.

For determining differences of temperature nothing equals this instrument, both for exactness of results and facility of application.

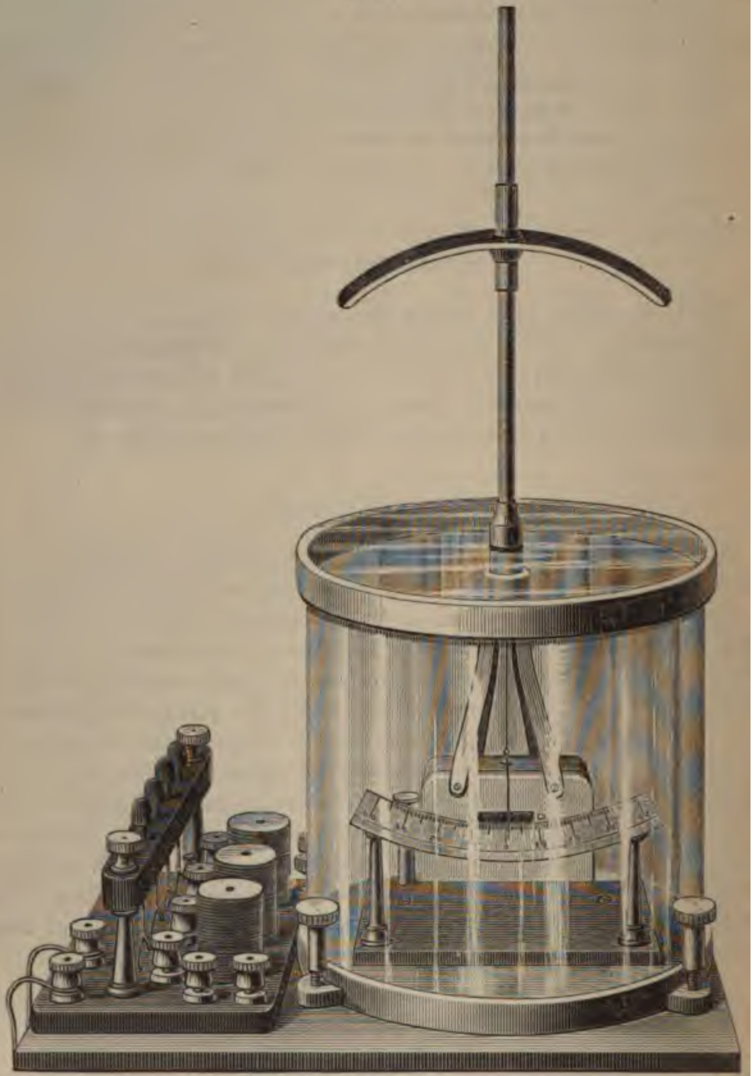
It consists, as shown in the accompanying cuts (Figs. 3 and 4), of

---

[1] *See* my "Memoir on the Pathology and Treatment of Organic Infantile Paralysis," in *Journal of Psychological Medicine*, No. 1, July, 1867, p. 53.

a galvanometer (Fig. 3) and two thermo-electric piles (Fig. 4). T
needle of the galvanometer is astatic, and is suspended by a delicate si

FIG. 3

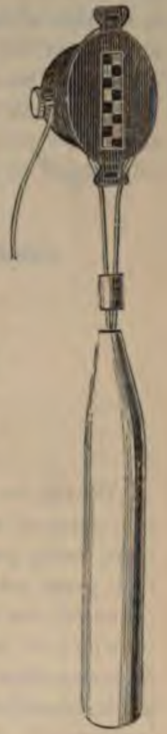


fibre so as just to swing clear of the scale it is to traverse. Above t
needle and outside of the glass shade is a magnet by means of whi
the needle is readily made to point to the zero of the scale.

Upon the ebonite plate to the left of the galvanometer need are the bobbins and four little cups of mercury by means of which the connections are made, and the resistance of the thermo-electric current increased or diminished, according as it is necessary to make the needle more or less delicate in its indications.

There are two thermo-electric piles; one of which is represented in Fig. 4, and which, for convenience of manipulation are furnished with handles. These piles are connected by their positive and negative poles, and the other positive and negative poles are connected with the stanchions seen on the ebonite plate of the galvanometer.

FIG. 4.



Having lowered the little metallic fork at the farther extremity of the bar over the ebonite plate, into the cup of mercury immediately under it, the apparatus is ready for use. The delicacy is increased by lowering one or two, or all three of the others, each one being in connection with the bobbin immediately opposite to it, and which, when the fork is out of the mercury, is included in the circuit, and hence has the effect of increasing the resistance. In the figure all the forks are represented as down.

To make an observation the thermo-electric piles are placed one on the part the relative temperature of which it is desired to know, and the other on the corresponding sound part. If the pile in connection with the stanchion nearer the corner of the ebonite plate is in contact with the hotter part, the needle will be deflected to the north. If the other be the hotter, the needle will be deflected to the south. The extent of the deflection indicates the relative difference in hundredths of a degree centigrade. It is to be remembered that the instrument must be placed on a firm table or stand, and must be so arranged that the end of the scale to the right of the cut points to the north; the ebonite plate will therefore be at the south end, and the galvanometer needle points to the east.[1]

With this apparatus of Dr. Lombard's it is easy to make relative determinations of temperature in a minute or two, and with great exactness and delicacy.

THE DYNAMOMETER.

Several forms of an instrument for measuring the strength of patients have been devised. The best and most generally applicable is that of M. Burq, modified by M. Mathieu, an instrument-maker of

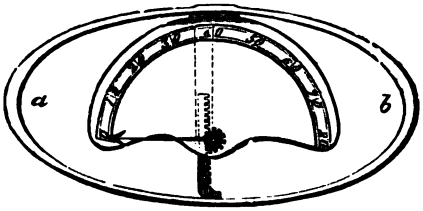
---

[1] For a fuller description of this instrument and directions for its use, the reader is referred to the *British Medical Journal* for 1875.

Paris. It is very simple, and for ascertaining the strength of th hands leaves nothing to be desired.

It consists, as is shown in the cut (Fig. 5), of an elliptical ste spring, to which is attached a semicircle of gilt brass, upon which scale is marked. An indicator, terminating at one end in a cog-whee is capable of being moved freely around the arc of the circle by a ste arm, upon one side of which, cogs, fitting into those of the indicato are cut. One end of this arm (the lower) touches the elliptical sprin when the indicator points to the zero of the scale ; a brass sheath upo the under side of the scale keeps this arm in place, at the same tim allowing it to move freely.

Fig. 5.



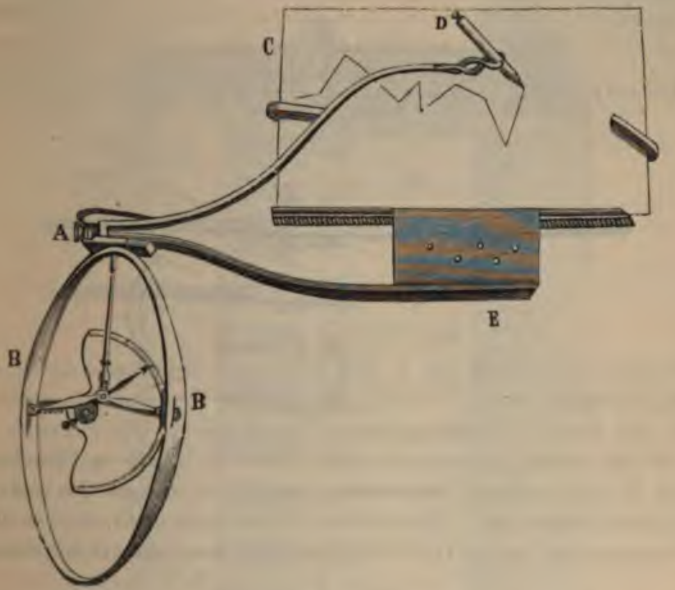
When the dynamometer is taken into the hand and pressed, t two sides of the spring are approximated, and the steel arm with th cogs, being pushed by the lower side of the spring, turns the indicato One great advantage of this instrument is that, when the pressure taken off, the indicator does not return to zero, but remains at the poi to which it has been carried by the muscular power of the individua We are thus enabled to see the extent of his strength, after he h made his effort, and do not have to watch him while he is using t instrument. It will also be seen that this dynamometer can be used measure tractile force; for, if two hooks with cords attached be fastene to the spring at the points *a* and *b*, traction on the cords will approx mate the two sides of the ellipse, and thus push the steel arm so as move the indicator as before.

THE DYNAMOGRAPH.

This instrument, which is of great value in the diagnosis of diseas of the nervous system, is shown in Fig. 6.

It consists of the dynamometer B B, to which a toggle-joint, mo ing a steel rod, is attached. This steel rod plays through a hole in t end of the elliptical spring and moves the lever which raises the pen D. At A is a screw which varies the point at which the rod touch the lever, and thus increases or lessens the delicacy of the indicatio C is a silvered plate upon which the paper is fastened by clips. To t lower part of this plate, a strip of gilt brass, with cogs cut in it, is attached. E is a gilt-brass box containing a watch-movement like that of the sphygmograph. A cog-wheel which projects above the upper side of this box fits into the cogs on the plate which carries the paper.

FIG. 6.



The wheel for winding up the clock-work, and the lever for stopping it or setting it in motion, are not seen in the figure, they being on the opposite side of the box.

To set the instrument in action, the sphygmograph movement is attached to the dynamometer at A. The clock-work is then wound up, and the plate holding the paper placed in the groove on top of the box E. The dynamometer is then grasped by the hand and squeezed firmly; the lever is thus moved, and the plate with the paper is carried along by the cog-wheel. As it moves, the pencil traces a line on the paper, the height and regularity of which depend upon the firmness and steadiness with which the dynamometer is pressed. As seen in the cut, the plate with the paper is in motion, and has about half completed its course. The patient should not look at the paper while using the instrument.

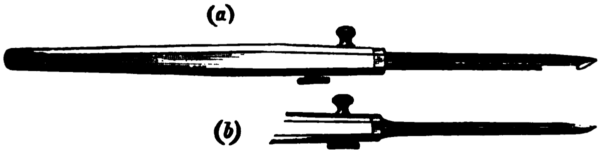
The dynamograph, therefore, writes down the muscular power and tone of the individual, and likewise indicates the perfection of what is sometimes called the muscular sense. A person in good health will make a straight line with the pencil. If there is paralysis of the muscles of the arm, or incoördination to the slightest possible extent, th line will be irregular. The papers used may be marked with the dat and the name of the patient, and thus a record of his condition is pr served.

The pencil should be of the very softest lead, and the paper shoul be rough and unsized.[1]

DUCHENNE'S TROCAR.

This very useful little instrument is shown in Fig. 7. It is intr duced open as at *a*. When it has perforated the muscle under exam nation, the small button at the under part of the handle is pushed fo ward; this propels a half-cylinder of steel against the shoulder at th end of the trocar, and thus a small piece of muscle is detached an caught in the cavity. The lower figure, *b*, represents the instrumer ready to be withdrawn. By drawing the button back, the bit of fib can be taken out, and is then ready for microscopical examination.

Fig. 7



ELECTRICAL APPARATUS.

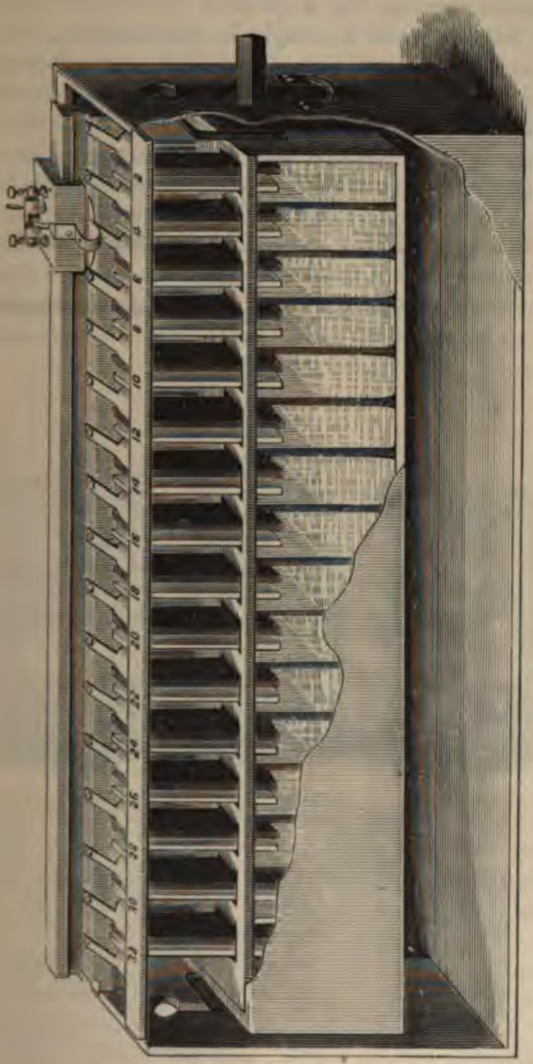
The electrical apparatus required in the diagnosis and treatment diseases of the nervous system must be of two kinds: one for furnisl ing the primary or galvanic current, the other for yielding the induce or faradaic current. Among the machines of the first category a those of Stöhrer, which are now made very satisfactorily by the Ga vano-Faradic Manufacturing Company of New York. An idea of th battery (thirty-two cells) will be obtained from the accompanyin woodcut (Fig. 8). The case is represented as broken away in front, s as to exhibit the internal arrangement. A combination of Smee's cel is manufactured by Dr. Jerome Kidder, and constitutes a good batter The very elegant battery constructed by Fleming and Talbot, of Phil delphia, is also very admirable in practice.

Some time since, my attention was drawn, in another connectio altogether, to the simplicity and efficiency of Hill's cell as a generat of a constant and most equable galvanic current. After an examin tion of its construction and action, I was satisfied that, for medical purposes, it was vastly superior to every other form of element which had come under my observation. At my suggestion, the Galvano-Faradic

---

[1] The first account of the use of the dynamograph was given by myself in the *Journ of Psychological Medicine*, January, 1868, p. 139.

Fig. 8.

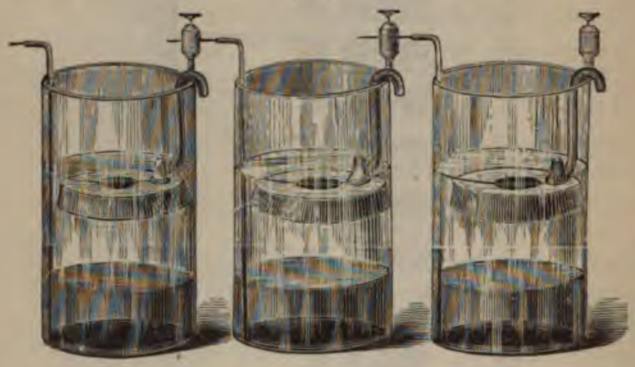


Manufacturing Company, of this city, has constructed, under the superintendence of Mr. Bartlett, a permanent battery, which, for office or hospital use, is of inestimable value. When erected it becomes, as its name implies, a permanent fixture. Several of these have been made one for myself, of one hundred elements, leaves nothing to be desire when used with Brenner's operating-table.

The cells used for this battery have been hitherto applied to telegraphy. They possess, however, in an eminent degree, the peculia qualities that are essential for a galvanic battery for therapeutic pu poses. The battery itself is simple in construction, easily managed exceedingly economical, utilizing almost all the materials consumed.

Each cell contains about half a gallon of fluid. A disk of sheet copper is laid flat on the bottom of the cell. To the under side of thi is affixed a copper wire, covered with gutta-percha. The copper shee forms the *negative plate;* the insulated wire which rises to the top o the cell, the *positive pole.* Two or three inches below the upper margin of the cell is suspended, by a brass hanger, a thick, disk-shape plate of zinc, concave on the lower side, with a round aperture in th centre. This is the *positive plate.* To the hanger is attached a bindin screw, and this forms the *negative pole.* Three cells *in situ* are represented in Fig. 9.

Fig. 9.



The body of the battery fluid is formed of a solution of sulphate o zinc. Occasionally, as required, crystals of sulphate of copper ar dropped through the central aperture in the zinc to the bottom of th fluid. These dissolve, and produce a layer of blue liquid, which cover the copper. Thus, we have copper in the bottom of the cell, immerse in a solution of copper, zinc suspended above, immersed in a whit liquid, the solution of zinc. (*See* engraving of these cells.) The mod adopted in other batteries to separate the fluids consists in using porous diaphragm, or cup, within, and surrounding which are place dissimilar metals and fluids. The porous septum, it was thought, woul allow the current to pass, and yet prevent the admixture of the divers elements. It has, however, been demonstrated that, when two suc liquids, and even two gases, are thus separated, they will invariably be come mixed. In this battery, without the intervention of any diaphragm, the denser liquid, the blue, remains in the bottom of the cell, the lighter one overflows and rests upon it; thus arranged, there is less liability to diffusion or mixing than if the two liquids were placed side by side, in vertical columns, with a porous partition between them.

The central aperture in the zinc plate also admits the introduction of a hydrometer to measure the density and strength of the liquid. Provision is also made for preventing too rapid evaporation of the fluid. The occasional addition of a little water, and every three or four weeks dropping in a few crystals of sulphate of copper, is nearly all that is required in the management of this battery. Further directions for its preparation, *modus operandi*, and care, may be obtained from the manufacturers.

Of faradaic batteries, those made by Kidder, the Galvano-Faradic Manufacturing Company, Fleming and Talbot, and others, leave nothing to be desired.

Magneto-electric machines—those in which the current is obtained by induction from a horseshoe magnet—are occasionally useful, and they have the great recommendation of being always ready and of not requiring a galvanic battery to start them. All that is necessary is to turn the helix by a crank. The objections to them are the facts that one hand must be employed in turning the crank, and that the strength of the current cannot be increased without, at the same time, increasing the rapidity of the interruptions. The current from such a machine does not cause anything like as much pain as that from a galvanic induction apparatus of like power.

Lately there has been a revival of statical electricity, and such perfect instruments for its production are being manufactured that this form promises ere long to come into very general use again. In a recently published paper, Dr. W. J. Morton very earnestly calls attention to the advantages of this form of electricity, or Franklinism, as it is properly called. I have witnessed some excellent results of its therapeutical power in cases of neuralgia and paralysis.

Although the applications of electricity in the treatment of diseases of the nervous system are not so extensively useful as asserted by some authors, it is nevertheless impossible for the physician to treat several affections of the kind mentioned without using the agent in some form or other. This is especially true of those diseases which are characterized by paralysis, in nearly all of which electricity is useful. In atrophic disorders, it is also indispensable, and in many hysterical conditions it is extremely valuable.

If only one battery can be procured, the faradaic instrument will be found more generally useful than any other; but, if possible, the physician who intends to treat to the utmost advantage diseases of the nervous system should possess one of all four kind mentioned.

CAUTERIZING APPARATUS.

It is often necessary, in the treatment of diseases of the nervou system, to make use of the actual cautery to the spine and other part of the body. The instruments formerly employed were very clums things made of iron, and, when required for use, heated in a furnac of some kind. The chief objection to iron is, that every time the in strument is heated to redness the surface becomes oxidized, and th scale may become loose, stick to the patient's body, or fall betwee his clothing.

Lately, instruments with platina tips of such shapes as may be r quired have come into general use, and are far preferable to those o iron. Being smaller, they are readily heated to redness or whitenes in a Bunsen's burner where there is gas, or in an alcohol blast-lamp.

Before using the cauterizing instrument, the skin should be ren dered anæsthetic by the ether or rhigolene spray. It is not necessar to carry the spraying to the point of congealing the skin, althoug there is no objection to so doing.

OTHER INSTRUMENTS AND APPARATUS.

Among the other instruments and apparatus required in the diag nosis and treatment of diseases of the nervous system are the micro scope, the sphygmograph, the stethoscope, ear-specula, tuning-fork urinary test apparatus and chemicals, hypodermic syringes, and Rich ardson's spray apparatus.

The latter is of use not only, as above mentioned, for renderin anæsthetic the part to be cauterized, but for refrigerating the skin i cases of chorea and other affections.

SECTION I.

DISEASES OF THE BRAIN.

CHAPTER I.

CEREBRAL CONGESTION.

CEREBRAL congestion is of two kinds, which differ as regards their mode of origin and symptoms. In the *active* form, there is an increase in the amount of arterial blood circulating in the vessels of the brain; in the *passive*, the quantity of venous blood is augmented. Occasionally the two conditions coexist.

ACTIVE CEREBRAL CONGESTION.

This is much the more common form. Of the cases recorded in my note-book, as occurring in my private and hospital practice, over five-sixths were of this description.

Andral, who, however, failed to distinguish the first or hyperæmic stage, recognized eight varieties, all of which may with advantage be comprehended in six, which are appropriately designated from the chief feature characterizing the attack, namely, the *apoplectic*, the *paralytic*, the *convulsive*, the *soporific*, the *maniacal*, and the *aphasic*, the latter being a sixth form, which is now for the first time systematically arranged in the present category. It will doubtless be the case that, as our knowledge of the functions of the brain becomes greater, other forms of cerebral congestion, especially those of a partial character—like the aphasic, for instance—will be recognized. Among these will be various sensory and motor disturbances, and perhaps also ab errations of mentality. For the present, however, it is perhaps better to defer considering these conditions, as often being instances of localized congestion, till the science of brain localization is more completely established.

Any of these may occur with scarcely a moment's warning. Generally, however, there is a premonitory or first stage, the symptoms of which, though well marked, are not peculiar, exclusively, to any one the fully established conditions mentioned. It is therefore impossib to predict with accuracy, from the symptoms of this prodromatic stag whether the apoplectic, the paralytic, the convulsive, the soporific, t maniacal, or the aphasic form, will be developed. An attentive stu of this stage should always be made, and active measures taken for t relief of the patient at a time when success can generally be obtaine

Symptoms. First Stage (Cerebral Hyperæmia).

Among the earliest symptoms of active cerebral congestion, wak fulness is especially noticeable, and may be for a time the only eviden of disorder which attracts the attention of the patient. He goes bed feeling weary, and as if sleep would very quickly overtake hi but he is disappointed, for he obtains but an hour or two of disturb slumber, which is generally broken by unpleasant dreams. Duri the remainder of the night he tosses restlessly from side to side of t bed, his mind either occupied by the thoughts which have occurred him through the day, or else filled with the most preposterous idea He consequently rises unrefreshed, feverish, and ill prepared for eith mental or physical exertion.[1] So far as the mind is concerned, the is an inability to give the attention to any subject requiring mu thought, and at times an absolute want of power to get correct ide of even simple matters. This is especially seen in those who have arit metical questions to solve, or long columns of figures to add up. I deed, mental labor of all descriptions is not only difficult, but is ir some in the extreme.

Before long the evidences of intellectual derangement become mo evident. The ideas are confused and without logical arrangemen the memory begins to fail, especially in regard to recent occurrence and there seems to be a special proclivity to forget words, and to su stitute others having a similar sound when pronounced, or appearan when written. The names of persons and places are particularly dif cult to recollect. The judgment is weak and vacillating; the mo strongly expressed determination is changed apparently without re son, and again there may be an impossibility of arriving at a decisi in cases where ordinarily but little reflection would be necessary. A effort toward continuous or severe thought increases the difficulties the mind, and augments the pain or uneasiness which generally exis in the head. Illusions, hallucinations, or delusions may be presen but are not usually fixed; and the patient will often laugh at t absurd images he has seen, or ideas he has entertained, not five mi utes before. Persons thus affected will frequently reason clearly regard to apparitions or voices, of the unreality of which they are full sensible.

---

[1] For a more complete account of wakefulness in all its relations, see the autho treatise on "Sleep and its Derangements." J. B. Lippincott & Co., Philadelphia, 1870.

A condition very often present is a *morbid apprehension of impending evil*, for which there is no assignable cause, and the nature of which the patient can rarely define. He is sure something will happen to him, but what, he does not know; or, if he does designate the form of trouble to ensue, he changes from one kind to another without any more reason than he had for the erroneous idea in the first place. Again, he is afraid that he may do some injurious act either against himself or others, and is hence fearful of being left alone. One patient was afraid to cross the ferry from Brooklyn lest he might be tempted to throw himself off the boat; another kept away from railway tracks, fearing that he might be led by the sight of a passing train to put himself in the way of the engine; another begged his wife to lock up his razors; and another would not take a warm bath, under the apprehension that he might neglect to turn off the hot water in time. It would be easy to enumerate very many more like instances. They remind us of "morbid impulse," but the subjects, unlike those of this last-named condition, never yield to the excitation. In fact, it is not an impulse, but the fear of an impulse, by which they are influenced.

The *emotional system* participates in the general mental disturbance, and indeed is often the part of the mind most prominently deranged. The passions are easily roused into activity by slight exciting causes; trifling circumstances produce great annoyance, and the little everyday troubles of life appear of vast importance. The disposition accordingly becomes suspicious, peevish, and fretful. Persons thus affected are very far, ordinarily, from being pleasant companions. Many of them avoid social intercourse, and shut themselves up in their rooms to brood over their real and imaginary disorders. Others, again, plunge into dissipation and excesses of every kind, in the vain expectation of being able by such means to overcome the disease; and again others strive, by a constant change of one physician for another, or the substitution of one quack medicine for another equally quackish, to get relief from their mental and physical distress. In some, there are very few decided symptoms present, except the inability to sleep, and the incapability of concentrating the mind upon an object of study or labor, without inducing pain or discomfort of some kind in the head.

In all, however, there is the same *mental introspection*. Every symptom is exaggerated; and, if one with which the patient has suffered should happen to be absent, he is dissatisfied till it makes its appearance again, or till he has, by concentration of his mind on the subject, brought it back, and with it an aggravation of all the other phenomena. "Doctor," said a gentleman to me a few days since, "I am afraid I am getting worse, for last night I slept several hours, and, if stupor should set in, I suppose it would be bad." Another, who had for several months suffered from an almost perpetual pain in the head, was quite sure sensibility was being destroyed when he found himself a whole day without it.

This fixing of the attention upon the body is of course apt to develop symptoms which would otherwise, doubtless, never make their appearance; and scarcely a day passes that instances in point do not come under my observation. The experiments of Mr. Braid in this direction are very instructive, and will bear quotation in the present connection. He requested four gentlemen, whose ages varied from forty to fifty-six years, and who were in good health, to lay their hands, palms upward, on a table, and to look at them fixedly for a few minutes. They were not to speak, but were, as far as possible, to concentrate the attention on the upturned palms, and to await the result. In about five minutes one of these gentlemen, a member of the Royal Academy, said that he felt a sensation of great cold in the hand; another, an author of distinction, said that at first he thought nothing was going to happen, but at last he felt a darting, pricking sensation, as if electric sparks were being drawn from the hand; the third, late mayor of a large city, reported that he felt a very uncomfortable sensation of heat come over the hand; and the fourth, secretary to an important association, had become rigidly cataleptic, the arm being firmly fixed on the table.[1]

Speaking of this subject, Sir Henry Holland[2] says:

"One limb, for instance, or even a single finger, or a portion of the sentient surface of the body, may be taken for observation, and the results tested and checked by means wholly independent of the subject of experiment, a point often very important to the truth of the result.

"We have here, as in other parts of the inquiry, to look to the respective cases of attention directed by express volition, or suggested by some outward cause acting on the mind. In the former and more simple case, if a limb be taken for experiment, a peculiar sense of weight with a vibratory tingling, or sensations approaching to cramp, are produced by the consciousness concentrated upon it. It is difficult to describe by words feelings of this nature, evanescent or changing at each moment, and different doubtless in different persons; but probably the closest resemblance is to those produced in ordinary cases by muscular fatigue or stagnant circulation through the limb. There is reason, indeed, to suppose that the muscular structure is actually affected in these cases, and frequently even by particular conditions of movement, though not volitional in kind."

Medical men are said, and doubtless with truth—as many cases will occur to the mind of the professional reader—to be particularly liable to be affected with the diseases to which they have given special attention; and every winter, during my course of lectures on the nervous system, I am consulted by medical students, who imagine themselves to be the subjects of the diseases I have brought to their notice; and in some cases with reason. Under another division of the subject, I shall have occasion to return to this matter for further consideration.

---

[1] For many illustrations of the power of the attention over the body, the reader is referred to the author's "Spiritualism, and Allied Causes and Conditions of Nervous Derangement." New York: 1876.

[2] "Chapters on Mental Physiology," p. 24. London: 1852.

It follows from what has been said, that, if well persons are liable to contract diseases through mental concentration, the subjects of cerebral hyperæmia must be peculiarly prone to the extension of their morbid symptoms through a like influence, and in fact this is exactly what occurs. A slight accidental sensation in some part of the body engages the attention, and becomes a fixture in the clinical history of the patient. Neuralgic pains, numbness, spasm, and even paralysis, may be thus induced, to say nothing of functional disturbances of the several organs.

Under this latter head there is none more frequently met with than what, for a want of a better name, may be called false impotence. To the production of this condition, the erroneous ideas which prevail relative to spermatorrhœa, and the fears excited by the advertisements and books of unprincipled quacks, largely contribute. Indeed, it is rarely the case that a male patient affected with cerebral hyperæmia does not at some time or other of its course imagine that he is impotent, and the only grounds he has for this notion are the facts that he has an occasional nocturnal emission, or the exudation of a little urethral mucus under the influence of sexual excitement. Still the fact is not to be overlooked that the predominance of this idea is extremely prejudicial to the patient's well-being, and it is therefore important that the physician should, by obtaining his confidence and enlightening his ignorance, dispel the delusion at the earliest possible moment.

In addition, there are certain physical symptoms of disordered cerebral action which by their prominence force themselves into notice. Thus there are *pain*, *heat*, a *feeling of fullness* or of *distention* in the head, the sensation as if a *tight band* encircled it, or the impression of a *dragging* or *clawing* character at the vertex. *Vertigo* is, however, the most prominent of all this category of phenomena in the majority of cases, and may be so severe as to prevent the patient moving about. In one case recently under my charge, the subject, a gentleman of about forty years of age, was often seized with intense vertigo while walking in the street, and was obliged at such times to seize hold of a lamp-post, or, if this was not within reach, to sit down on the nearest door-step, or even the curbstone, till the violence of the attack had in a measure abated.

Again, the least movement of the body, the slightest attempt at mental exertion, or the most trifling emotional disturbance, is sufficient to excite it. At times it is clearly aggravated by indiscretions in diet or the ingestion of even a small quantity of any stimulating liquor, and at others is present during the whole period of being awake. There are two kinds of this vertigo. In one the patient seems to be in motion ; in the other the objects about him appear to be tumbling topsy-turvy around him. In the latter the ground in front appears to rise up to meet him, and hence he walks as if ascending a hill. In some cases the two conditions coexist or may alternate. Probably no symptom is more distressing than this. It almost invariably excites more fear of serious consequences than in reality should attend it, and it prevents the patient taking that bodily exercise so conducive to his restoration to health. In some cases, however, it is entirely absent, though such are, I think, rarely met with, and, no matter how intense it may be, is scarcely ever accompanied by nausea.

In other cases *headache* constitutes the chief physical feature of the disease, and even when not predominant is a more or less constant attendant on the morbid condition. It may be very severe, unfitting the sufferer for the slightest mental or physical exertion, or may consist of a dull, aching pain, very wearing, but yet bearable. It is aggravated by any effort to use the mind or body, and especially by any cause—such as a dependent position of the head, the use of stimulating ingesta, a constriction about the abdomen, chest, or neck—likely to increase the amount of the intra-cranial blood.

In some cases there is no actual pain, except as the immediate consequence of some one or other of the existing causes mentioned ; but the patient is always conscious of an uncomfortable sensation in the head, which, if not a pain, is capable of being readily converted into one. This is, as I have said, sometimes a mere feeling of fullness or tightness, or as if the brain—so a patient described it—were "being gathered together into a heap," or, as another said, were "being scratched with a claw." Again, there is the impression that the head is exactly balanced on a very sharp point, and that some effort is required to keep it from falling off.

Usually the painful sensations in the head disappear toward night, or on the attempt to sleep, but resume their violence as soon as the patient awakes in the morning.

The special senses could scarcely be expected to escape giving evidences of derangement, and hence among the chief manifestations of the intra-cranial disorder are those connected with the perceptive organs.

Thus there are noises in the ears, such as roaring, rumbling, or singing, and occasionally loud reports, such as might be produced by the discharge of fire-arms. A gentleman, recently under my care for the affection in question, informed me that when he first experienced the sensation mentioned he was sitting in his library, quietly reading, when he suddenly heard a report as if a pistol had been shot off within a foot of his head. He jumped to his feet, expecting to see an assailant behind him, but, to his surprise, there was no one to be seen, and it was very evident that no explosion had taken place. He was greatly astonished at this, but attributed the whole matter to an exaggeration, excited by his irritable nervous system, of some street noise. He had no further experience of the kind till the following morning, when, on rising from bed after a wretched night of sleeplessness, he again heard the sound, and this time it was as nearly as possible like the noise produced by striking two stoutly bound books together close to his ears. After this there was scarcely a day that the sound was not heard. It was entirely subjective, as persons in close proximity to him at the time heard nothing.

Several such cases have come under my observation. It is not in all that the sound appears to be in the ears. In some it has seemed to be located in different parts of the head, generally, however, in the posterior region.

In some cases patients have experienced the sensation as if something snapped or gave way within the head, and this has, in a few rare instances, been attended with the sudden disappearance of some of the more striking symptoms. Thus, a young lady, in consequence of an intense emotion, was seized with sudden vertigo and pain in the head, and fell to the floor unconscious. Recovering her senses in a few minutes, she found herself unable to speak a word, though she uttered in an excited way inarticulate sounds having no resemblance to speech. This condition continued for several hours, when she suddenly felt "something snap" in the head, and she instantly recovered the power of talking. The vertigo, pain in the head, and other symptoms, persisted for two or three months afterward.

In another case the onset of the disease, in a gentleman who had for many years overworked his brain, was extremely sudden, and was attended with facial paralysis. I treated him for this latter condition with electricity, with but little benefit; but one day he struck his head violently against a gas-burner hanging over his desk, and shortly afterward felt something give way within his head with a sharp, snapping sound, and the paralysis instantly disappeared, after having lasted some five or six days.

Such cases are, in the present state of our knowledge, inexplicable.

The ear becomes hyperæsthetic, and loud noises are therefore disagreeable. At times the sense of hearing is morbidly acute, while at others it is markedly impaired. Sounds are misinterpreted with some persons, and illusions result. This is especially the case at night, when the patient is lying awake, the mind stretched to its utmost tension. A gentleman informed me that a circumstance with which most persons are familiar—the conversion of the sound of the ticking of a clock into some phrase or other—was to him a matter of agonizing weariness. Night after night as he lay in bed, the ticking of a large clo in the hall seemed to be the constant repetition of the word "fa well." Not wishing to reveal the matter to others, he endured f many nights the consequent suffering, till finally he made an excuse f leaving the city. But still the wheels of the railway cars seemed to uttering the word "farewell," and it was only after a fatiguing journ to Baltimore and repose in a quiet room that he escaped the inflicti

In addition to illusions, hallucinations of hearing are not unco mon, and are usually in the form of whispered words, which t patient hears with as much vividness as though they were real utt ances. Like the misinterpretations of real sensorial impressions, the are usually experienced at night, and may be excited by any circu stance, mental or physical, which tends to increase the amount of blo circulating in the intra-cranial vessels. Thus, a powerful emotion, unusually severe mental task, a strong muscular effort, or a depende position of the head, may induce them. In one case, that of a gentl man of rather obese development, a whisper of some kind or oth was always heard when he stooped to button his gaiter-boots. another, straining in the water-closet frequently caused a like sym tom. In one very interesting instance the sounds were like those musical instruments, and were arranged into familiar tunes, to the small satisfaction of the subject; and in another they assumed t similitude of the bark of a dog. Occasionally they are in the fo of commands to perpetrate some act of violence, such as suicide. patient, who came from Brooklyn to consult me, heard a voice wh pering in his ear, and ordering him to throw himself into the riv "What is the use of your going to see a physician?" it said. "T best thing you can do is to kill yourself. You are of no service yourself or any one else. Jump overboard and end the matter once." Though these hallucinations never imposed upon the reas of the patient, they were nevertheless sufficiently distressing, givi rise, as they did, to the fear that he might, some day or other, be i fluenced by them to commit an act which he abhorred.

The aural speculum is almost, if not quite, as valuable as the op thalmoscope in affording important information relative to the aff tion under notice; and I have been in the habit for the last five yea of employing it in every case presenting the more obvious features the disease. I do not mean to be understood as intimating that po tively affirmative results are to be obtained in all instances, but neith are they of any other single symptom. That the tympanum do afford an indication of the state of the intra-cranial circulation sufficiently evident, from a consideration of the experiments perform by my friend Prof. Roosa and myself[1] relative to the influence of t sulphate of quinine, the results of which have been amply confirmed by the subsequent investigations of Prof. Roosa, as well as by those of other observers.

---

[1] "The Influence of the Disulphate of Quinine over the Intra-Cranial Circulatio *Psychological and Medico-Legal Journal*, October, 1874, p. 230.

In the cerebral disorder under notice, evident congestion will almost always be observed of the vessels over the handle of the malleus, and the tympanum will be seen to be of a light pinkish color. In some cases we are prevented making the usual examination owing to the accumulation of cerumen. This must be removed by forceps or by washing, and the inspection deferred till next day.

I may add that physicians, wishing to observe the connection between cerebral hyperæmia and tympanic congestion, have a ready method of satisfying themselves on this point by examining the tympanum before and after the subject has inhaled a few drops of the nitrite of amyl. This was first done, so far as I am aware, by Mr. Galton,[1] and detailed in his paper entitled "Notes on the Condition of the Tympanic Membrane in the Insane."

The *faculty of vision* is almost invariably more or less disturbed. Sometimes there are bright flashes of light, from over-excitation of the retina, and these, like the other symptoms, are rendered more intense upon mental or physical exertion. At other times dark spots—*muscæ volitantes*—render the vision indistinct; and again there is the appearance of an undulatory vapor, such as is seen around a hot stove, or on a plain heated by the sun. The conjunctivæ are suffused; the pupils contracted. There is intolerance of light, and motion of the eyeballs is painful, and the ophthalmic symptoms are aggravated by the effort to use the eyes. The ocular muscles easily become fatigued, and hence pain is excited by any attempt to read or to adjust the visual foci for near objects.

Ophthalmoscopic examination shows the arteries of the retina to be increased in size and tortuosity, and vessels which in health are not visible are now clearly perceived. The optic disk is often more or less congested, exhibiting the appearance to which Allbutt has applied the name "Congestion Papilla," but which is perhaps more generally known as "choked disk." The tint of the choroid is deeper than it is when in a normal condition.

The effect of cerebral congestion in giving rise to visual hallucinations has long been known, though it often happens that in practice the value of the fact as an indication of the state of the intra-cranial circulation is in a great measure disregarded. In another work[2] I have considered the subject of hallucinations of sight at some length, and, as showing the influence of undoubted cerebral congestion in producing them, I quote the following case which occurred in my own experience:

---

[1] "West Riding Lunatic Asylum Medical Reports," vol. iii., 1873, p. 258.

[2] "Spiritualism and Allied Causes and Conditions of Nervous Derangement." New York, 1876, p. 8.

"A gentleman under the professional charge of the writer c
always cause the appearance of images by tying a handkerchief mod
ately tight around his neck, and there is one form which is alwa
the first to come and the last to disappear. It consists of a male figu
clothed in the costume worn in England three hundred years ago, a
bearing a striking resemblance to the portraits of Sir Walter Raleig
This figure not only imposes on the sight, but also on the hearing ; 1
questions put to it are answered promptly."

"A similar instance is related in 'Nicholson's Journal.'[1] 'I knov
gentleman,' he states, 'in the vigor of life, who, in my opinion, is n
exceeded by any one in acquired knowledge and originality of de
research, and who for nine months in succession was always visit
by a figure of the same man, threatening to destroy him, at the ti
of his going to rest. It appeared upon his lying down, and instant
disappeared when he resumed the erect position.'"

A case somewhat like the first of the two foregoing is referred
by De Boismont,[2] in which an individual was able to obtain hallucin
tions of sight by inclining his head a little forward. By this mov
ment, the return of blood from the interior of the cranium was pr
vented, and hence a state of repletion favorable to the production
hallucinations was induced.

Now, in the state of cerebral hyperæmia which results from exc
sive brain-work or intense emotional disturbance, a condition exis
not essentially different from that present in the case referred to, e
cept in the circumstance that the excess of blood is mainly arteri
instead of venous, and that hence the congestion is more active th
passive. But it must be borne in mind that it requires a very gre
degree of hyperæmia to cause the production of visual hallucinatior
and therefore that we are not to expect them to occur in all patien
who are its subjects. So far as my own experience extends, only abo
one in five exhibits the symptom with any degree of distinctness.

Double vision is occasionally a phenomenon of the disease in que
tion, though it is generally transient, and, as Krishaber remarks, ord
narily only manifested in regard to bright objects.

This author also speaks of a peculiarity of sight which has n
come under my notice. "A patient," he says, "looks at himself in
glass with astonishment, as if he had forgotten his appearance. A
other is horrified at his image, which represents a being altogether
different traits from those which he conceived himself to possess. B
he is not alarmed, for he knows that it is only his perception which
changed. This aberration exists not only as regards his own perso
but other objects as well. The patient finds men and things changed

---

[1] Vol. vi., p. 166.

[2] "History of Dreams, Visions, Apparitions, etc." American edition. Philadelphi
1855.

he is astonished, always astonished, and it seems to him that he is a being transported to another planet."[1]

The *sense of smell* is very often lost, perverted, or intensely exalted. Perhaps the second named of these changes is the one most frequently met with. I have a patient now under my care, a gentleman, who from over-mental work is suffering from cerebral hyperæmia, and who constantly, while awake, smells the odor of illuminating gas. So strong is this, that he is at times unable to resist the impression that gas is escaping somewhere, and he goes from burner to burner of his residence and office seeking for the imaginary leak. Another is constantly sensible of the smell of turpentine or new paint, and another has the odor of mint constantly present in his nostrils.

The *taste* is also occasionally affected in like manner, usually, so far as my experience goes, in the way of perversion. "Things don't taste as they used to," is a common complaint, and the saliva and buccal mucus often give the gustatory impression of other substances. It is not at all unlikely, however, that "the bad taste in the mouth," so often mentioned by patients, is due to a real change in the properties of the saliva or mucus. I have observed several cases in which any mental or emotional strain was sufficient to cause a bitter or other unpleasant taste in the mouth, and the same phenomenon is quite common as a consequence of gastric disturbance. Krishaber cites two cases in which both smell and taste were entirely abolished.

Sensation and the power of motion are usually affected, and generally, though not always, on one side of the body only. Thus, the arm or the leg feels heavy, and a feeling as of ants crawling over it, pins and needles sticking in it, or as if the limb were "asleep," is experienced. Sometimes these sensations are confined to the face, the muscles of which feel drawn or tight, and the skin of which has the various indications of anæsthesia mentioned. Most frequently, however, they are, I think, experienced on the scalp, giving rise to the several sensations already mentioned.

Again, there is an exaltation of the sensibility of the skin and of the sensory nerves generally, and thus neuralgic pains are felt in various parts of the body; or the cutaneous surface is extremely sensitive to the impression made upon it, whether of heat, cold, or slight pressure.

Slight convulsive actions or twitchings of individual muscles or groups of muscles are generally present. Sometimes a few fibers only are affected. The face, and especially the eye-lids and angles of the mouth, is particularly liable to be thus involved. The muscular strength is usually weakened. The patient tires after slight physical exertion, and occasionally certain muscles, such as the deltoid and tibialis anticus, become distinctly paretic, so that there is an impairment of the ability to raise the arm from the side or to elevate th foot sufficiently high in walking to clear ordinary inequalities in th pavement. The dynamometer shows the grasp of the hand of one c other side, or of both, to be weakened, and the line made by the dynamograph is zigzag or uniformly depressed.

---

[1] *Op. cit.*, p. 168.

The *appetite* is capricious, and the *stomach* acts imperfectly an sluggishly. The gastric juice is not secreted in sufficient quantity fo the purposes of digestion, and, the peristaltic action of the stomac being weakened, the food remains within it a long time undigeste and undergoing fermentation. Regurgitations, both of the solid contents and of gases, are common, and the patient tastes his meals several hours after they have been swallowed. Gases accumulate in th stomach, and give rise to the sense of fullness experienced even after very slight repast has been taken. Such symptoms are usually classe under the name of "nervous dyspepsia," a not improper designatio if it does not lead us into the error of regarding them as of primar importance, instead of considering them, as they are, merely consequen on the head trouble.

The *bowels* are ordinarily costive, though at times this conditio alternates with diarrhœa.

The *urine* is in some patients scanty and high-colored, in others i is profuse and almost as pale as water. Oxalate of lime is often present, and an excess of phosphates an invariable condition, so far a my experience extends. I have already spoken of this circumstanc Whether or not the phosphates in the urine are to be regarded as th ashes of the nervous system, and hence a measure of the amount o nerve tissue decomposed, there is no doubt that they are inordinatel increased after intense mental or emotional strain.

I have spoken of the heat of the head of which the patient generally complains. That there is a real increase of temperature can ofte be perceived by the hand or by the use of an ordinary thermomete But in some cases the actual rise of temperature is so slight, notwithstanding the feeling of heat which the patient experiences, that we cannot detect it by either of these means. In such cases resort should b had to the thermo-electric differential calorimeter of Lombard, b which very minute changes of temperature can be detected, and th part of the brain in which the temperature is highest be readily ascertained. The experiments of Lombard, performed several years ag show very beautifully the influence of cerebral action in augmentin the external heat of the head, and it may be remembered that, ove two years ago, I detailed to the Neurological Society the results o some experiments of my own in the same direction. For several yea past I have never examined a patient presenting the more obviou features of cerebral hyperæmia without carefully determining the surface temperature of various parts of the scalp. At times and in som regions the elevation reaches two degrees of centigrade above the normal standard.[1]

But one of the chief categories of symptoms remains to be considered—chief, at least, so far as the more obvious appearances go, though, like the other visceral derangements, I must regard these as being due to the brain disorder—and that is the group of phenomena connected with the heart. To Krishaber, in the work already cited, belongs the credit of being the first to call attention to this remarkable series, for in the publication of my own, to which I have referred, it was in a great measure overlooked. As Krishaber remarks, the troubles of the circulation consist especially in an irritability of the vascular system, so that the least movement, such as rising erect from the sitting posture, or to the sitting from the recumbent, leads to an acceleration of the pulse of from 20 to 30 or even 40 beats a minute. Besides this, there are frequent and violent palpitations, either spontaneous, or provoked by the most insignificant causes, either mental or physical.

Emotional excitement is, however, the most prolific cause of cardiac disturbance in patients affected with cerebral hyperæmia, and at times leads to serious results. The pulsations of the heart may be so irregular and the action of the organ so strong as to induce grave interference with the respiratory apparatus. Upon one occasion a lady, while in my consulting-room, was seized with a paroxysm of the kind in question, of so severe a character that for a moment or two I thought she was about to die. For several months she had been wakeful, had suffered from vertigo and slight pain in the head, and, while relating to me her symptoms, a blast near by, where a cellar was being excavated, exploded, and produced so violent and sudden a shock as to bring on the excessive cardiac action mentioned. The heart throbbed with so great a degree of violence that its pulsations could be readily seen through her dress and heard at the distance of two or three feet; her face and neck became livid, and, gasping for breath, she fell to the floor insensible. In a very short time, however, the inordinate movements ceased, and she recovered consciousness.

Physical examination of the heart fails in these cases to reveal the existence of any organic lesion.

In the intervals between the paroxysms of inordinate cardiac action, the pulse is small, often slow, soft, compressible, but by r means regular, either in force or frequency. Intermissions of tl beats are a common phenomenon, and give rise to anxiety and morbi apprehensions in the patient.

---

[1] Since the above was written I have become acquainted with some recent experiments of Prof. Broca, of Paris, in the same direction. As he does not refer to either Lombard's or my own experiments, though the former ("Experiments on the Relation of Heat to Mental Work") were published in the *New York Medical Journal*, January, 1867, p. 198, and a synopsis of my own in the *Journal of Nervous and Mental Disease*, January, 1876, I presume he is unacquainted with either. Prof. Broca ascertained by means of thermometers, applied to different parts of the scalp, that the external temperature was affected by different internal morbid and physiological conditions, and hence confirmed the previous observations of Lombard and myself. His experiments would have yielded much more delicate and accurate results if he had employed Lombard's instrument.

Krishaber states that at the very beginning of the disorder the is sometimes present a series of phenomena simulating fever, suc as a chill, followed by a distinct period of febrile excitement. Du ing this last stage the temperature of the body is elevated almo half a degree centigrade, or nearly a whole degree of our scale, an may even be double this. This accession may be repeated with son degree of periodicity, but it soon ceases, and does not reappear aft the full development of the disease.

I have observed this condition in about one third of the cas that have come under my observation, though usually close questio ing is necessary to elucidate the fact of its existence, so little in pression does it make upon the mind of the patient. Sometime however, the paroxysms are of such severity as to excite the beli that they are of malarious origin, and, being treated with quinin they and the other symptoms attendant on the disease are greatl aggravated.

During the most intense period of the disease there are occasio ally paroxysms characterized by entire inability to move a muscle (the body, the consciousness, respiration, and circulation not being m terially disturbed. I have never had a case which exhibited the symptoms, though Krishaber appears to regard them as not uncommo On the other hand, syncope with complete loss of consciousness, whic he speaks of as rare, is, according to my experience, by no mea uncommon. With both of these conditions, there is an almost co tinuous præcordial pain, sometimes severe enough to excite the ide of the existence of angina pectoris, and causing the gravest appr hensions on the part of the patient and his friends.

That one of the primary effects of intellectual exertion or em tional disturbance is an increase in the amount of blood circulatin through the brain, does not admit of a doubt, except from those wh still refusing to learn, contend that the cerebral circulation is not sul ject to variation under any circumstances. Experimental physiolog has, however, determined this point so positively in the affirmativ that it is scarcely necessary to adduce the evidence in its support. I will be sufficient to recall the numerous facts observed by others an myself with reference to the immediate cause of sleep, by which it i shown that during the condition of wakefulness the quantity of bloo in the brain is much greater than it is during sleep, the first being state of intellectual activity, the latter one of almost complete cer bral rest.

Excessive mental exercise inordinately augments the activity c the cerebral circulation. The blood-vessels become over-distended, and, if the brain be kept long in a condition of extraordinary action, they may be rendered incapable of returning spontaneously to their normal dimensions. Like a bladder filled to repletion with urine, they become in a manner paralyzed and unable to contract upon their contents. They lose, to a certain extent, their elasticity, and, like the India-rubber band kept too long around a large bundle of papers, they do not regain their natural size even when the distention is removed. A state of cerebral hyperæmia is thus induced, which gives rise to a set of perfectly characteristic symptoms, and which is fraught with peril to those in whom it occurs.

In a monograph published some seven years ago, Dr. M. Krishaber[1] described a disorder of the brain and heart which is probably identical with the one under consideration, and to which, under the name of cerebral hyperæmia, or the prodromatic stage of cerebral hyperæmia, I called attention in the first edition of my "Treatise on Diseases of the Nervous System," published in 1871. Krishaber's studies have very considerably advanced our knowledge of the subject, and, as my own more recent investigations and enlarged experience have tended still further to the elucidation of a very interesting and important condition of the nervous system, I have thought it would not be out of place to bring some of the more notable results of our labors to the notice of the Neurological Society. It may be as well, however, to state here, at the outset, that I differ with Krishaber entirely relative to the pathology of the disorder we have both described, and that I am of the opinion that the cardiac symptoms upon which he lays great stress are really of quite secondary importance. In other respects there is no essential point of difference between us in the representations of an affection studied independently of each other, and from altogether different standpoints.

The disease is sometimes developed with great suddenness, but ordinarily it advances little by little to completeness. When the former is the case, the patient experiences, under the influence of great mental excitement, pain in the head, vertigo, an inability to speak, or, at least, imperfection of articulation. There are noises in the ears, flashes of light before the eyes, and occasionally for a short time double vision. The heart beats with increased force and rapidity, and is more or less irregular in its action; the face is flushed, and a feeling of suffocation is experienced. If he attempts to walk, his gait is uncertain or staggering, not only in consequence of the vertigo present, but from actual loss of power in the limbs. Numbness is commonly felt in some part of the body, and clonic spasms of the muscles, notably of those of the face, are generally present.

With all these physical symptoms, there are others indicating mental disturbance. Chief among these are hallucinations, or illusions of the senses, particularly of sight and hearing. Insomnia is an almost invariable attendant, and what little sleep the patient obtains is interrupted by unpleasant or even frightful dreams. Gradually the disorder becomes established, and then other functions, especially those connected with digestion, are deranged. From the first the urine is loaded with urates and phosphates.

---

[1] "De la névropathie cérébro-cardiaque." Paris, 1873.

As instances of the suddenness with which the disease may make its onset, I cite the following cases from my note-book :

F. H., a gentleman engaged in a manufacturing business which required all his attention to make it profitable, was informed one morning by his superintendent that a large lot of material had been spoiled. He at once experienced an intense sensation of vertigo, a sharp pain in the head, palpitation of the heart, and would have fallen, had he not been supported by the bystanders. There were also a roaring sound in the ears and flashes of light before the eyes. On attempting to stand, the vertigo and palpitations were increased. There was at no time loss of consciousness, though the ideas were confused and the speech thick. In the course of a few hours the severity of these symptoms diminished, but that night he was unable to sleep, and in the morning the morbid phenomena reappeared, though with diminished violence. For several months afterward he was troubled with wakefulness, a sense of fullness and tightness in the head, occasional weakness of the limbs, slight numbness, and a total inability to exert his mind in his business affairs without an increase in all the symptoms. Under appropriate treatment he entirely recovered.

S. L., a book-keeper, after a day of unusually arduous work, left his place of business to go home. He had hardly taken half a dozen steps when he was seized with vertigo, and fell unconscious on the sidewalk. He almost immediately regained his senses, but, on trying to stand, found that he was paralyzed in both legs, and that the least motion of the body brought on a return of the vertigo, which was now attended with pain in the head, mostly in the frontal region, noises in the ears and indistinctness of vision. On attempting to speak, his articulation was so imperfect that he could scarcely be understood. There was an uneasy feeling at the pit of the stomach, but neither nausea nor palpitation of the heart, though the action of this organ was irregular. He was taken home in a carriage, and after a sleepless night found himself very little better, except in the fact that, though his legs were still weak, there was no absolute paralysis. Gradually he got somewhat better, though walking always produced vertigo, and his gait was similar to that of a partially drunken man, as he found it impossible to avoid a zigzag course, or a decided tendency to sidle over to the edge of the pavement. Sleep was almost every night imperfect, being disturbed by dreams of difficulties from which he could not extricate himself, such as the house being on fire, and, on his springing from bed, discovering his door to be locked on the outside ; falling into the water, and being on the point of drowning from inability to divest himself of heavy boots, and so on. Mental application was impossible without leading to an aggravation of all his symptoms, and the least emotional excitement was sufficient to augment them to a high degree. He suffered in this manner for nearly a year, before relief was obtained, being in that time treated with remedies directed to the removal of cerebral anæmia, when, in fact, the intra-cranial condition was directly the opposite.

M. S., a young lady, aged nineteen, and without notable predisposition to neurotic disturbances, was deeply chagrined at not being invited to a ball at which she had confidently anticipated being present. While talking the matter over with some friends, she suddenly experienced a severe pain in the head, vertigo, noises in the ears, flashes of light alternating with darkness, and violent palpitation of the heart. At the same time a peculiar thrilling sensation was felt throughout the body, especially on the left side. These symptoms continued with great intensity all that day, notwithstanding that stimulants and antispasmodics were administered in large quantities by the physicians called to attend her. During the night, every attempt to turn over in bed was attended with vertigo and palpitation of the heart. For over a year there was very little improvement, and the course of the disease was not essentially different from the other cases cited. The most distressing symptom in her case was the persistence of the insomnia, it rarely happening that she obtained over an hour or two of unrefreshing slumber. When she came under my care, some thirteen months after the inception of the disease, I found that the affection, though mitigated in the violence of the attendant phenomena, was still sufficiently distressing to impair her capacity for enjoyment and her usefulness to others. Recognizing the existence of congestion of the brain rather than anæmia, for which she had uniformly been treated, I acted accordingly, and had the satisfaction of seeing her gradually improve, till, at the end of less than six weeks, she was as well as she ever had been in her life.

These cases are cited, not as exhibiting perfect representations of cerebral hyperæmia, but merely for the purpose of illustrating the suddenness with which the condition may be induced. They are selected at random from many others occurring in my hospital and private practice, and detailed in my note-book.

Eventually, no matter how brusque may be the development of the symptoms, the course of the disease is not materially different from that of the more gradually established form next to be described. Indeed, there are no differences except as regards the order of sequence in which the symptoms ensue and in the fact that, in the present form, there is, in the beginning, a greater degree of intensity in the abnormal manifestations.

In the majority of cases, therefore, the affection is evolved more slowly, and the order of appearances of the phenomena somewhat different.

The foregoing constitute the ordinary assemblage of symptoms which are first met with in congestion of the brain. Some of them may be absent, others so slightly manifested as to escape ordinary observation, and others, again, so strongly exhibited as to excite the grave apprehensions of the patient and his friends, and to require him to keep his bed. Generally, however, they are not so severe as to prevent him attending in a measure to his ordinary avocations, and they may altogether disappear, either spontaneously or in consequence of appropriate medical treatment.

A spontaneous cure is, however, rare, and, without proper management on the part of the patient or his medical attendant, the symptoms pass, sooner or later, into one of the fully developed forms mentioned. Thus, of the cases that have been under my observation, the disease was arrested at the first stage in about ninety-five per cent. by appropriate treatment, while there was not a single instance of spontaneous cure.

The fact that abscesses of the liver may be associated with cerebral hyperæmia, probably as a direct result, was pointed out by me[1] a short time since, and several cases detailed in which aspiration had led to the evacuation of pus from the liver. Since the publication of the original paper on the subject, other similar cases have come under my notice, and like ones have been reported by other observers.[2] It is probable, however, that other brain lesions—as is well known of blows upon the head—are capable of inducing the condition in question. The subject will be more appropriately considered in detail in my forthcoming work on mental disorders.

SECOND STAGE. *a. The Apoplectic Form.*—Occasionally this variety of cerebral congestion is initial, but ordinarily it is preceded by the group of symptoms just detailed. In either event the onset is generally sudden. The patient is perhaps walking in the street, when he staggers, loses consciousness, and falls. The loss of intelligence and sensibility is, however, rarely complete, and may last but a few minutes or even seconds, though sometimes continuing for several hours.

Paralysis, to a greater or less extent, is always present for a time.

---

[1] "On Obscure Abscesses of the Liver: their Association with Hypochondria and their Treatment." *St. Louis Clinical Record*, June, 1878.

[2] "The Diagnosis of Abscess of the Liver by Symptoms of Cerebral Hyperæmia," etc. By J. Marion Sims, M. D. *Virginia Medical Monthly*, January, 1880.

"Hyperæmia of the Brain associated with Hepatic Abscess," by W. H. De Witt, M. D. *Medical Gazette*, April 3, 1880.

One limb only may be affected, or those of one side, or all four members. It is never complete, the patient being able to perform some movements, though not to exert his full strength. The face is rarely involved, and the patient, though answering briefly when addressed in a loud voice, speaks indistinctly and with difficulty. The respiration is loud, slow, but rarely stertorous, and it is not often that there is puffing of the lips and cheeks. The pulse is slow, hard, and full. Sometimes the face is flushed, and sometimes it is unusually pale. The sphincters generally retain their power. The senses, though weakened, are often capable of being exercised by tolerably strong excitations. A bright light causes uneasiness and closure of the eyelids. A loud noise is productive of discomfort, and a limb, when pinched, is withdrawn. The power of the mind is greatly lessened, and some faculties are altogether abolished. Answers, more or less direct, are given to simple questions put in a loud tone, but even moderate intellectual action seems to be impossible.

Gradually the attack passes off, leaving the patient in a state of mental and physical depression, which may last for several days. The paralysis usually disappears, but occasionally it does not, one or more limbs or muscles remaining permanently, or for a long time, disabled.

It sometimes happens, however, that the termination is not so favorable. The vessels may remain congested, serum may be effused, and death may result without there being any vascular lesion. Two cases have come under my notice, in which death ensued from this cause in first attacks.

A person who has once had a paroxysm, such as has been described, is thereby rendered more liable to subsequent seizures, each one of which still further permanently impairs his mental and physical powers. In one case, occurring in my practice, there have been eleven attacks in five years; and in another, fourteen in four years. In both of these, and in several similar instances I have witnessed, there was paralysis, which had become more profound with each accession. It is therefore inexact to say, as do some writers, that the paralysis of cerebral congestion always disappears in a short time.

The apoplectic form of cerebral congestion is more common than any other of the fully developed varieties, about one half of all the cases being of this type.

b. The Paralytic Form.—Like the apoplectic variety, this may be unpreceded by the premonitory symptoms constituting the first stage, but usually they have been present. The loss of power or of sensibility, or of both, may be very circumscribed, limited to a single group of muscles in the one case, or a small portion of the cutaneous surface in the other, or one entire side, or both sides of the body, may be involved. It differs from the apoplectic form in no essential respect, except that there is no loss of consciousness. Its onset is sudden.

c. The Convulsive Form.—This, like the variety just described, may come on suddenly, or may be preceded by premonitory symptoms. The phenomena of the attack do not generally differ from those attendant on an ordinary epileptic paroxysm, except that there is never an aura, and no peculiar cry, such as is so often met with in pure epilepsy. There is the same tonic spasm, followed by clonic convulsions, which may or may not be confined to one side of the body, and which may or may not be followed by temporary or long-continued paralysis. Stupor likewise supervenes, but is neither of so long a duration nor so profound as in true epilepsy.

In other cases, and especially in infants or young children, there is no loss of consciousness. The pain in the head is intense, the pupils are contracted and insensible to light; there are vomiting and acceleration of the pulse. The convulsive movements, which may be either tonic or clonic, or both alternately, are either quite general or confined to a single limb or even a group of muscles.

This form of cerebral congestion is never developed during sleep, for then the brain contains less blood than when the individual is awake. It may occur during stupor induced by certain drugs, constriction of the neck, or a dependent position of the head; but stupor is not sleep, although the two conditions are frequently confounded. Convulsions occurring during ordinary sleep are never the result of congestion. This point will be more fully considered under the head of epilepsy.

After the stupor the patient may feel comparatively well, or there may be delirium, continuing for several hours. As in the apoplectic form, there may be a succession of attacks, and the mind and physical power of the patient are thereby greatly weakened.

The variety under consideration is, perhaps, more liable to occur in individuals past the age of forty, though I have witnessed several cases in quite young persons. It is not often met with in old age, and, when it is, is generally fatal, probably from secondary lesion. A majority of the cases of epileptiform convulsions, occurring for the first time in persons over the age of forty, are instances of the convulsive form of cerebral congestion.

d. The Soporific Form.—This form will be more fully described under the head of passive cerebral congestion, to which condition it is almost entirely restricted. It differs from the apoplectic form in the circumstance that the invasion is gradual; and from this and the paralytic in the fact that there is no paralysis, although the limbs may be in a state of general resolution. The chief phenomena are pain in the head, dilatation of the pupils, and stupor.

e. The Maniacal Form.—This variety, though not so common as either of the others, is yet not infrequent. It is characterized by an accession of mental derangement not materially different from that indicative of acute mania. The delirium is of a very active character, th eyes are suffused, the face is red, the head hot, the motility active, and the whole manner, character, disposition, and mental processes are changed. During the paroxysm, the patient may commit some act of violence, and it almost always happens that his combative proclivities are aroused. He may likewise attempt to injure himself.

The attack may come on with great suddenness. In the case of a gentleman recently under my charge, it was the result of eating a hearty meal in a great hurry at a railway station. A few minutes after his return to the train, he was attacked with furious delirium, during which he attempted to injure himself and all within his reach. He was seized and held, but continued, as far as he was able, to bite, scratch, and kick at those who were near him. The paroxysm lasted about two hours. He then fell into a heavy stupor, from which he did not arouse for two hours longer. For several days his mind was weak, and there was numbness in various parts of his body. Gradually, however, he regained his former powers, but he suffered from occasional confusion of thought and difficulty of speech, with headache and wakefulness, for several weeks.

In another case—that of a boy thirteen years of age—it was characterized by paroxysms of maniacal excitement, during which the subject attempted to bite and otherwise to injure those around him, indulging at the same time in the most profane and obscene language. These seizures took place about once a week. There was generally a distinct recollection of all the events which had happened. In several other cases, the seizures were the result of malarial poisoning, and were exactly periodical in their occurrence. Paralysis, as in the other forms, may be one of the phenomena of this variety of cerebral congestion. Death may take place during the attack, or from secondary lesions afterward.[1] What is called temporary insanity, mania ephemera, or impulsive insanity, generally depends upon cerebral congestion. The subject, therefore, is of vast importance in its medico-legal relations.[2]

f. The Aphasic Form.—The inception of this type is usually very sudden. There may or may not be the accompaniments of pain in the head, vertigo, and confusion of mind. The chief symptom is the impairment or abolition of the faculty of speech, and this may be the only phenomenon. A very interesting case is that of Prof. Lordat, which is graphically described by Trousseau.[3] The loss of speech was at first complete, but was entirely regained in twelve hours.

---

[1] The whole subject of cerebral congestion has been well considered by Calmeil, in his "Traité des Maladies Inflammatoires du Cerveau." Paris, 1859.

[2] See a memoir by the author, entitled "A Medico-Legal Study of the Case of Daniel McFarland," in the *Journal of Psychological Medicine* for July, 1870; also published separately by D. Appleton & Co. New York, 1870. Also a paper on "Morbid Impulse," *Psychological and Medico-Legal Journal*, August, 1874.

[3] "Lectures on Clinical Medicine," etc. Translated by P. Victor Bazire, M. D. London, 1866, p. 219.

Several similar instances have come under my observation. In a case at this time under my charge, the patient, a lawyer, was suddenly deprived of all power of speech, after passing several hours in very intense study. There was a little confusion of ideas, but neither pain nor vertigo. There was loss both of the memory of words and of the power of so coördinating the muscles of speech as to articulate. There was no paralysis anywhere. Recovery was complete in less than six hours.

In two cases occurring in my own practice, the patients were suddenly rendered aphasic by inhalation of the nitrite of amyl. The effect continued for half an hour in one case, and for nearly an hour in the other, after all the other phenomena from the amyl had entirely disappeared.

The subject of aphasia will be more fully considered in a subsequent part of this work.

It is quite probable that certain disturbances of the sensory organs, restricted spasmodic actions, and paralyses, illusions, and hallucinations, intellectual, emotional, and volitional impulses of a morbid character, and other abnormal cerebral manifestations, to some of which attention has been directed, are the results of localized and quite limited congestions of the brain. As already said, however, it would be premature to differentiate these with any attempt at exactness till our knowledge of the various sensory, motor, and mental centers of the brain is more exact than it is at present.

THIRD STAGE.—This period may be considered as beginning after the immediate effects of the paroxysm, whether it has been of the apoplectic, paralytic, convulsive, maniacal, or aphasic form, have passed off. It is characterized by feebleness of body and mind, by gastric or intestinal derangement, by pain in the head, with transient attacks of vertigo, and occasionally by numbness and slight paralysis of one or more of the limbs. Many of the symptoms met with in the first stage are again found in this.

But the principal phenomena are those connected with secondary lesions, such as inflammation, abscess, softening, and adventitious growths of various kinds. These will be considered under their proper heads. It must not be forgotten that one circumstance always exists, and that is, the proclivity to other paroxysms of some one of the fully-developed forms.

PASSIVE CEREBRAL CONGESTION.

This condition is the result of causes which increase the amount of venous blood in the brain. It is more commonly met with in old persons and in those of feeble constitution. Women are more frequently affected than men.

Symptoms. FIRST STAGE.—As in active cerebral congestion, there is a premonitory stage, the symptoms of which are similar to those previously described. There is, however, a tendency to stupor, and the other phenomena are, in the main, less strongly marked. Vertigo, pain, illusions, hallucinations, and delusions, are nevertheless generally present at one time or another. But the stupor, or tendency to somnolence, is the most prominent feature, and the sleep, even when comparatively natural, is attended with dreams, unpleasant or even frightful in character.

The degree of congestion may be suddenly increased, or, what is a more probable sequence, there may be effusion of serum, and then in either case the second stage, exhibiting itself as in the apoplectic, the paralytic, the convulsive, the soporific, the maniacal, or the aphasic form, results.

The proportion of cases of passive cerebral congestion which pass to the second stage is greater than in the active form of the affection, and it is accordingly a more serious disease.

SECOND STAGE. *a. The Apoplectic Form.*—In this variety the onset of the affection is sudden, like that of active cerebral congestion. The loss of consciousness is generally complete, the face is red, the pupils are dilated and insensible to light, the respiration is stertorous, and the fæces and urine may be passed involuntarily. The action of the heart is slow and feeble, and the pulse corresponds to these facts. Paralysis may be general, or confined to a lateral half of the body.

If sensibility returns, there are pain in the head, vertigo, tinnitus aurium, generally some embarrassment in the speech from lingual paralysis, and more or less loss of the power of motion in other parts of the body. There will also be general or partial anæsthesia. As the condition of the patient improves, these symptoms generally disappear. Death, however, is not an infrequent sequence. This form of cerebral congestion is most common with elderly persons, and appears to be particularly apt to attack old women.

b. The Paralytic Form.—This does not differ essentially from the apoplectic form, except that there is no loss of consciousness, the paralysis constituting the main symptom. It may be either sudden or gradual in its inception.

c. The Convulsive Form.—This may not differ materially from the convulsive form of active congestion, except as regards increased length of the fit and prolonged stupor. Generally, however, there is a repetition of the seizures, and I am led to believe, from my experience, that there is a greater tendency to biting the tongue. Paralysis is a more common sequence, and is of longer duration, and the mind appears to suffer more seriously and at an earlier period.

d. The Soporific Form.—The first symptom observed is commonly a general numbness and indisposition to muscular exertion. The drowsiness, which has probably been present to some extent, increases, and

soon becomes the most notable feature. At first, it is easy to rou
the patient from this stupor, but it gradually becomes more profou
and overpowering, until at last a persistent comatose condition
reached. The faculties of the mind may, in the earlier stages, be e
cited into a moderate degree of activity ; but with the advancing con
they are no longer capable of being manifested. The cutaneous sen
bility becomes less and less, the urine dribbles, from paralysis of t
bladder and its sphincter, and the bowels, if not obstinately constipate
allow their contents to pass involuntarily. With these symptoms, t
pupils are dilated, and, as long as sensibility exists, pain in the head
complained of. The faculty of speech is impaired at an early peri
but, although the tongue is restrained in its movements, there is
actual paralysis of this or any other muscle. This condition may la
for several weeks, and, though recovery occasionally takes place, this
never complete. Death is the more usual termination.

e. The Maniacal Form is not often met with in passive cerebr
congestion, and, when it is, the delirium, so far from being of a furio
type, is low. The patient mutters to himself incoherently, and exhibi
great muscular restlessness, but never attempts to do violence to hi
self or others. Coma often occurs as a sequence.

f. The Aphasic Form.—Aphasia without other complication is n
often met with as a consequence of passive cerebral congestion. T
instances only have come under my notice, and in both the developme
was much slower than is usually the case in the active form of the aff
tion. In both of these there was disease of the right side of the hea
manifested by mitral and tricuspid regurgitation, jugular pulsatio
great fullness of the veins of the neck and face, and ascites and gener
anasarca. The loss of the idea of language was complete in both case
and persisted for about forty-eight hours. There was no paralysi
stupor, or convulsion, and but slight pain. The ophthalmoscope r
vealed the existence of great turgescence of the retinal veins, with v
nous pulsation.

Causes.—The causes of cerebral congestion are : of the active form
those influences which are capable of increasing the quantity of arteri
blood in the brain : of the passive, those which produce a similar eff
upon the amount of venous blood circulating in the vessels within t
cranium. The causes of the first category induce activity of circulatio
those of the second torpidity.

The causes of active cerebral congestion may either, by their gra
ual operation, initiate the premonitory stage, or they may sudden
induce the development of this stage into one or other of the varieti
already described as constituting the second stage. Among them
temperature either very high or very low. Thus, the disease is mo
frequent in hot climates than in those of more temperate character, an
in the summer months than in the spring or autumn. It is, howeve

more common in very cold than in warm weather. Thus Andral, of one hundred and fourteen cases, found that twenty-six occurred in summer and fifty in winter. My own experience is to the same effect, as will be seen from the following table, which embraces the cases in my private practice in the city of New York during a period of five years, beginning January, 1865, and ending December, 1870 :

January	66	July	68
February	64	August	74
March	50	September	27
April	39	October	31
May	42	November	52
June	37	December	72
Total			622

An examination of this table shows that one hundred and ten cases occurred in the autumn months, one hundred and thirty-one in the spring, one hundred and seventy-nine in summer, and two hundred and two in winter. All my subsequent experience is to the same effect.

Passive cerebral congestion is very much more frequent in cold than in warm weather.

The direct rays of the sun are capable of producing sudden attacks (insolatio), of which congestion is a prominent feature, but which require separate consideration ; and it is not uncommon for artisans, whose heads are exposed to heat from furnaces, to suffer in a similar manner.

Some authors contend that certain winds increase the liability to cerebral congestion. Leuret, quoted by Mosmant,[1] could attribute an epidemic of cerebral congestion, which appeared at Charenton, to nothing but a long-continued wind from the northwest. The supposition that atmospheric electricity is a causative influence rests upon nothing but hypothesis.

Unhealthy situations, such as those subject to the influence of malaria and to noxious emanations of any kind, and which are not well ventilated, also predispose to attacks of cerebral congestion.

The ingestion of a large quantity of food into the stomach may occasion passive congestion, by the pressure which the distended organ makes upon the large veins of the abdomen. Rapid eating, even though the quantity of food be moderate, may cause the active form of the affection by some influence exerted through the sympathetic system.

Sudden and violent physical exertion, especially if made in the stooping posture, is very liable to induce cerebral congestion. Childbirth is an instance in point, and I have known several cases to be caused by severe straining in the water-closet. The constipation of the bowels rendering such efforts at defecation necessary is itself productive of the disease.

---

[1] "Essai sur la Congestion Cérébrale." Paris, 1858.

A dependent position of the head and constriction of the neck from the dress are also, by impeding the return of blood from the head, liable to induce congestion of the passive form.

Certain articles of food and medicine, such as spices, alcoholic liquors, opium, belladonna, quinine, etc., act either by augmenting the power of the heart, or by their effect on the sympathetic, paralyzing the vaso-motor nerves, and thus increasing the caliber of the cerebral blood-vessels. In this connection, the influence of the nitrite of amyl, when inhaled to increase the quantity of blood in the brain, may be cited as an instance of this latter power.

Tumors in the neck, or in other parts of the body where the return of blood from the head may be impeded by their pressure, likewise cause congestion. Other causes are to be found in certain diseases, as fevers of various kinds, erysipelas, disorders of menstruation, the suppression of hæmorrhagic or other discharges; local affections of the brain, as embolus, thrombosis, tubercle or apoplectic clots, and sympathetically by worms in the intestinal canal, or irritation existing in other portions of the system. Hypertrophy of the left side of the heart is a common cause of active cerebral congestion; and any affection of the right side of this organ, tending to impede the return of the venous blood, is an important factor in giving rise to the passive form of the affection under notice.

But the most influential and common causes of cerebral hyperæmia, and eventually of congestion, are to be found in long-continued intellectual exertion, mental anxiety, or sudden, violent, or prolonged emotional disturbance. It is from the action of such factors that the premonitory symptoms are generally induced, though they may, especially those embraced in the last-named category, immediately develop a fully formed attack. The fact that cerebral exercise increases the amount of blood in the head is made evident to all of us at times, by the distention of the superficial vessels, the suffusion of the eyes, the heat and pain which we feel when we have overtasked our brains. Cerebral action is always attended with hyperæmia, just as is the activity of the liver, the kidneys, or other organs. Active cerebral congestion is thus induced, and is, within certain limits, perfectly normal. But these limits are liable to be exceeded—and, in this active period of the world's history, often are—and then the condition described as the first stage of congestion is established. The vessels, from continued over-distention, lose their contractility, just as I have said does the India-rubber band, used to keep a bundle of letters together when the package is too large, or it has been kept stretched for a long time. An additional disturbing force, heat, cold, an overloaded stomach, increased mental labor, emotional excitement, or any of the causes mentioned, may suddenly evolve a fully developed paroxysm.

Emotion acts in a similar manner, though, as has been said, often with more suddenness. The emotions of shame, of anger, and others cause the face to become red from dilatation of the blood-vessels, and a like effect is produced in the vessels within the cranium. If the emotion is very strong or lasting, a correspondingly increased hyperæmia results.

There are certain circumstances which render the action of the causes specified more effectual or powerful. These are inherent in the individual, and may be classed as predisposing causes. Among them are sex, the disease being more common in males; age, it being more frequently met with in middle-aged or old persons; hereditary influence; hypertrophy of the left ventricle of the heart, by which the flow of blood to the head is directly increased; dilatation of the right ventricle, by which its power is diminished, and the return of blood from the head impeded; insufficiency of the auriculo-ventricular valves, or constriction at the auricular or ventricular orifices on the same side, by which a similar result is produced, and perhaps, though this point is by no means established, shortness of the neck.

Diagnosis.—Cerebral congestion may be confounded with cerebral hæmorrhage, meningeal hæmorrhage, embolism, thrombosis, softening, epilepsy, urinæmia, stomachal vertigo, auditory vertigo, and with the very opposite condition, cerebral anæmia. From each of these affections it is, however, distinguished by well-marked characteristics.

The premonitory symptoms are not liable to be mistaken for cerebral hæmorrhage, but this error may be made as regards the second stage. The apoplectic form is, however, distinguished from apoplexy due to extravasation, by the fact that in it the loss of intelligence is rarely complete, and that, when it is so, the mind is dormant but for a few moments; that sensibility and the power of motion are never altogether abolished; that coma, when present, is rarely profound; that the paralysis, when it exists, is seldom limited to one side of the body; by the general absence of stertor, and puffing of the lips and cheeks in breathing; and by the short duration of the symptoms.

From meningeal hæmorrhage, it is discriminated by the comparative lightness of the symptoms, and by the fact that they do not progressively augment in severity or intermit in violence.

Cerebral congestion and embolism present some features in common, and it is therefore occasionally difficult to distinguish them. In the former, however, the pulse is slow and the respiration regular and deep; in the latter, the pulse is more rapid, is often irregular, as is also the respiration; in the former, there is increased heat of the head; in the latter, the temperature of this part of the body is unchanged; in cerebral congestion the symptoms are transient; in embolism they are more lasting; in the former there is often a distinct premonitory stage; in the latter, the attack always takes place without a moment's warning. In the former, though there may be cardiac difficulties, they are different from those predisposing to embolism, which are consecutive to endocarditis—generally rheumatic—and which implicate the semi-lunar or mitral valves, and in the fact that recovery from an attack of cerebral congestion is generally complete, which is rarely the case in embolism.

From thrombosis, cerebral congestion is diagnosticated by the circumstances that in the former the progress of the disease is slow, that there is usually well-marked paralysis from the beginning; that the phenomena indicating mental disturbance are more strongly pronounced; that the articulation and memory for words are more permanently affected; and, notwithstanding occasional remissions, by the persistency and gradual advance of the symptoms.

In softening there are often a sudden loss of consciousness, persistent hemiplegia, and death in a few days. Again, there is delirium without paralysis or convulsions, and in other cases there is a gradual accession of the symptoms. This latter is the only form liable to be mistaken for cerebral congestion. It is attended with headache, feebleness of intellect, and a gradually advancing paralysis generally, beginning in one of the lower extremities, and extending to the whole of one side of the body. The speech is always seriously impaired, and the mental disorder is of a far graver character than that due to cerebral congestion. The gradual advance of the affection to a fatal termination is also a characteristic circumstance.

With urinæmia, cerebral congestion may be confounded, if only the more obvious head symptoms be taken into consideration. The history of the case and full inquiry will always, however, enable the proper discrimination to be made. Thus, in urinæmia the existence of kidney disease, as evidenced by a chemical and microscopical examination of the urine, the anasarca of the face or limbs, and the repeated attacks of convulsions and coma, will be sufficient diagnostic marks.

From epilepsy, cerebral congestion is distinguished by the fact that the former is not preceded by the group of symptoms constituting the first stage of congestion; that the congestion of the vessels of the face and neck is preceded by a death-like paleness; that an aura is often present; that there may be a peculiar cry; that the patient does not stagger and fall slowly to the ground, but drops as if knocked down by a severe blow; and that the tongue is frequently bitten. The reverse is the case as regards all these phenomena in cerebral congestion. Nevertheless, so accurate and experienced an observer as Trousseau in his clinical lecture on "Apoplectiform Cerebral Congestion in its Relations to Epilepsy and Eclampsia,"[1] confounds the two conditions.

---

[1] "Clinique Médicale," tome ii., p. 56. Also Bazire's Translation, London, 1866, p. 19.

Trousseau's views on this subject do not, however, appear to be accepted by any large number of medical authorities. Epileptic vertigo is, as will be shown at a proper place, a very different affection from any form of cerebral congestion, and is not likely to be confounded with it. Epileptic mania has, likewise, very few points in common with the disease under consideration.

In stomachal vertigo the attacks of dizziness are often severe, but they are clearly associated with gastric derangement, and only occur while the stomach is digesting its contents. Other symptoms of dyspepsia will also be noticed, while the mental and physical disturbances, which constitute so prominent a feature of cerebral congestion, are absent. The distinction, however, is not always made.

In auditory vertigo, or Ménière's disease, the dizziness is accompanied with aural troubles, such as deafness and tinnitus ; the face is pale ; and there is almost invariably vomiting, or at least intense nausea. Moreover, when there is loss of consciousness, the premonitory symptoms are not such as precede the second stage of cerebral congestion, but are connected with the function of audition.

From cerebral anæmia, the first stage of congestion is frequently not clearly distinguished, and I have seen several cases in which patients had been treated for the one condition when the other was indubitably present. In both there are headache, sense of constriction, vertigo, noises in the ears, numbness, mental confusion, loss of memory, inaptitude for labor of any kind, and at times loss of consciousness. But in anæmia the face is not flushed, the carotid and temporal arteries do not throb with violence ; the pulse is quick, feeble, and irregular, the respiration is hurried, the pupils are dilated, there are bellows murmurs at the base of the heart and in the veins of the neck, and the general aspect of the patient is not of that rugged appearance so generally associated with cerebral congestion. In the syncope of cerebral anæmia, the paleness of the face, coldness of the skin, and feebleness of the heart's action, will serve to draw the line between it and the apoplectic form of congestion. The ophthalmoscope will at all stages prove of great value in the diagnosis.

Prognosis.—The prognosis is materially modified, according to the stage of the disease present when the patient is seen, and the form of attack from which he may be suffering. Active cerebral congestion is a more favorable type than the passive. If the affection has not gone beyond the first stage, a fortunate issue may safely be predicted under the use of suitable medical treatment ; but, if, through neglect or improper treatment, or indiscretion on the part of the patient, the disease becomes fully developed, the prognosis is much more grave. I have never known a death to take place in any patient from this disease during the premonitory stage. The apoplectic and soporific forms are the most grave, and the prognosis is rendered more unfavorable with each attack. The epileptic form is ordinarily not dangerous to life, nor is the paralytic, maniacal, or the aphasic, except in old persons. Occasionally, however, even in young and robust patients, death ensues during the paroxysms of these forms.

The liability to secondary lesions, such as softening, cerebritis, hæmorrhage, aneurisms, general paralysis, etc., must be taken into account when forming a prognosis. The more frequent the paroxysms of any form, the greater the risk of some such finality.

The habits of the patient are also important elements in forming an opinion in regard to the ultimate result. If these are bad, and are persisted in, the probability is that no treatment will be of much avail in preventing a recurrence. Moreover, by such a condition of the brain as the excessive use of alcohol, inordinate mental exertion, or continual emotional excitement induces, the chance of escaping some secondary morbid process is very much lessened.

Of the one hundred and seven fully developed cases which have been under my observation during the past eight years, there were eighteen deaths; seven from the apoplectic form, all after repeated attacks; three from the maniacal, one of which was that of a young man about thirty years of age; and seven from secondary lesions. Of these latter, four were from softening, one from cerebritis, one from hæmorrhage, and one from general paralysis.

Morbid Anatomy.—There are certain appearances seen in the brains of those who have died of cerebral congestion which are characteristic, although it must be confessed that some or all of them are occasionally absent. These are:

An increased size of the capillaries and large blood-vessels, both of the brain and the pia mater. It thus happens that, when a section of the brain is made, the red points ordinarily seen are larger and more numerous than usual, and that the pia mater presents in spots, or throughout its extent, a red or rose-colored appearance.

The white matter of the brain is increased in consistence and density, and the gray matter is red, or even violet in hue.

There is sometimes a large quantity of subarachnoidean effusion, the ventricles may contain an excessive amount of fluid, and the choroid plexuses are often enlarged.

If there have been repeated attacks of cerebral congestion, it is not unusual to find, by microscopical examination, little granules of hæmatin in contact with the blood-vessels. The same means of exploration shows the minuter capillaries to be more than naturally tortuous, and to have little aneurismal swellings. These may or may not involve the whole circumference of the vessel. Their presence and import were first pointed out by Laborde.[1]

---

[1] "La ramollissement et congestion du cerveau principalement considérés chez le vieillard." Paris, 1866.

On making a transverse section of the hemisphere, a cribriform appearance is seen, if the patient has repeatedly suffered from attacks of cerebral congestion, and especially if he be advanced in years. This is due to the presence of numerous little holes with sharply-defined margins. The brain-tissue bounding these is generally without material change, either in color or consistence. This condition, called by Durand-Fardel,[1] to whom the credit of first describing it is usually given, "l'état criblé," is supposed to be due to the fact that the vessels have been so distended during life as to press with increased force upon the perivascular tissue, and that, shrinking after death, they no longer fill their former space, which remains empty. Calmeil[2] was the first to notice this condition. He has very often found, in maniacs, the white substance rendered cribriform by vessels distended with blood, sometimes empty, but always greatly dilated. This state, although frequently met with in congestion, is not uncommon in other pathological conditions, such as the several forms of softening, of which, however, congestion is often the first stage.

Durand-Fardel[3] calls attention to the fact that, on making sections of the medullary substance of the cerebrum, it is not uncommon to find in cases of congestion rose-colored patches scattered throughout its substance. On examining these with a lens, they are seen to consist of a large number of delicate vessels partially injected. I have never witnessed this appearance, except in one instance, nor is it noticed by authors on the subject generally.

If the congestion has been severe or long continued, the convolutions may be to a considerable extent obliterated by the compression of the brain against the internal wall of the cranium. At the same time, the membranes of the brain are rendered dry and viscous from the pressure to which they have been subjected.

In passive congestion the sinuses of the dura mater are the chief seats of vascular turgescence; the veins generally are distended, and there is ordinarily a greater amount of serous effusion in the subarachnoid space than in the active variety of the disease.

Pathology.—It is almost useless at this day to discuss the question of the possibility of the quantity of blood in the brain being subject to variation. Still, it may be interesting to recall briefly the facts which establish the affirmative in the matter.

In the cases of infants, in whom the anterior fontanelle is still open, the scalp is seen to be elevated above the level of the skull when the head is dependent, and depressed when the head is elevated.

The same fact is observed in persons who have suffered injury of the skull, involving the loss of a portion of its substance. During strong emotional excitement, or the action of any cause capable of increasing the force of the circulation, the scalp is elevated. From the action of opposite causes it is depressed. Both in infants and in persons who have received injuries such as those cited, the scalp is seen to be depressed during sleep, and to rise as soon as the individual awakes.

---

[1] "Traité pratique des maladies des vieillards." Paris, 1854, and deuxième édition, 1873.

[2] "De la paralysie considérée chez les aliénés," etc. Paris, 1826.

[3] *Op. cit.*, Paris, 1873, p. 21.

A dependent position of the head causes a sensation of fullness, or even pain, and blood may flow from the nostrils. The eyes are observed to be "bloodshot," and the countenance indicates congestion. A tumor, a ligature, or any other cause capable of exerting pressure on the jugular veins, will produce like effects. Ophthalmoscopic examination under such circumstances shows the veins of the retina to be enlarged, indicating that an obstruction exists to the return of blood through the sinuses and veins within the cranium. Post-mortem examination of persons dying, who, during life, have suffered interruption to the perfect return of blood from the head, reveals the existence of intracranial congestion. Animals, subjected to experiments calculated to act in the manner stated, are after death found to have congested brains.

In animals bled to death the brain is found anæmic to an extreme degree.

Direct experiment still more positively establishes the fact under consideration. If a portion of the skull of an animal be removed, and the aperture be then securely closed with a watch-glass, the vessels will be seen to enlarge and contract according to the cause brought into action, and the brain will be correspondingly elevated or depressed.

By means of an instrument, devised, independently of each other, by Dr. S. Weir Mitchell and myself, the degree of pressure within the cranium can be accurately measured. It is thus seen that the quantity of blood circulating in the brain undergoes material variation.[1]

The anatomical arrangement of the blood-vessels of the cerebral tissue is such as to admit of an enlargement of their calibre without necessarily subjecting the perivascular substance to pressure. Robin[2] discovered the existence of sheaths around these vessels, and his observations were subsequently confirmed by His,[3] who ascertained that the same arrangement exists in the spinal cord. According to His, "Fine transverse sections of a hardened brain, having its vessels injected or otherwise, show that all the blood-vessels, arteries, veins, and even capillaries, are surrounded by a clear space, broadest in the case of the larger vessels, but in all cases quite sharply defined externally. In transverse sections the vessels are seen to be surrounded by a ring-like space, and in parallel sections the space is seen on each side of the trunk of the vessel, and follows it in all its ramifications."

---

[1] For a more complete argument on the subject, and for a statement in detail of the experiments of Mr. Durham and myself on this point, the reader is referred to the author's monograph, "Sleep and its Derangements." Philadelphia: J. B. Lippincott & Co., 1870. The cephalo-hæmometer referred to in the text is described in that work (Appendix), and also in the introduction to this treatise.

[2] *Journal de la physiologie de l'homme et des animaux*, 1859, p. 527.

[3] "Zeitschrift für Wissenschaftliche Zoologie," 1865, B. xv., quoted in the *Journal of Anatomy and Physiology*. Translation by Dr. Bastian.

These perivascular canals are lined by a hyaline membrane, and are capable of being injected, and, in cases of chronic congestion, may become permanently enlarged, so as to cause the appearance referred to under the heading of morbid anatomy.

The pathology of the subject receives further elucidation from a consideration of the causes capable of giving rise to cerebral congestion, and which have been already mentioned in detail.

Treatment.—Recollecting the two grand forms of cerebral congestion, the principles which should guide us in treatment will be clearly apparent. In the active type of the disease, the force of the cerebral circulation and the quantity of blood in the blood-vessels of the brain are to be lessened; in the passive variety, the force of the circulation is to be increased, and at the same time the accumulation of blood in the veins to be diminished. In the active form of this affection, the abstraction of blood from the arm was formerly very generally practised, but is now rarely perfomed. I have never seen a case in which it was required. Local bleeding is more generally applicable, and a few cups to the nape of the neck will often afford marked relief. Leeches to the temples are also useful, though they are preferably applied just inside the nostrils. I have many times witnessed the most satisfactory results from a couple of leeches thus used, and from accidental nasal hæmorrhage.

The application of the actual cautery to the nape of the neck is also a measure of value, especially in the earlier stages. It is preferable, I think, to any other form of counter-irritation, and, when properly done, is not at all painful. It seems to have a positive and, in some cases, an immediate influence in diminishing the calibre of the cerebral arteries.

Cold is another very useful agent in the treatment. It may be applied to the nape of the neck, or directly to the cranium, either as very cold water or in the form of ice.

The advantages of position should also be brought to bear. The head should be kept elevated, especially during sleep, and no severe muscular exertion should be taken while stooping.

The clothing should be kept loose about the neck. As a derivative, a mustard-plaster applied to the epigastrium is often of service; and the same may be said of warm or even hot water to the feet. Blisters I rarely employ, though I have occasionally done so with advantage.

The constant galvanic current possesses the power of contracting the cerebral blood-vessels, when so used as to stimulate the sympathetic nerve. For this purpose, one pole should be placed over this nerve in the neck, and the other on the back of the neck, as low down as the seventh cervical vertebra. The current from about fifteen Smee's cells is sufficient, and it should not be allowed to act for more than two minutes. If extreme vertigo be produced, the number of cells should be lessened. This property of the primary current was first pointed out by Bernard, Waller, and Budge, but its demonstration by the ophthalmoscope was first made by myself. Observation with this instrument, while the current is acting, shows that the vessels of the retina contract, and hence there can be no doubt that the result is produced upon those of the brain. A similar effect is caused by passing the current directly through the brain, the poles being applied to the mastoid processes. A slight feeling of vertigo follows both when the circuit is closed and opened. The good effects of this practice are well marked, a few applications being often sufficient to abolish the vertigo and unpleasant feelings in the head, and to restore mental and physical activity.

Of internal remedies the number is not large, and those which it is advisable to employ are generally effectual, with or without the external measures mentioned, in entirely relieving the patient.

First among these must be placed the bromide of potassium. Several years ago I pointed out the value of this medicine, and explained the *rationale* of its action. As others have since claimed the discovery as their own, I hope I may be excused for quoting the following passage from a memoir upon an analogous subject,[1] in which the action of the bromide is clearly indicated :

"Bromide of potassium can almost always be used with advantage to diminish the amount of blood in the brain, and to allay any excitement of the nervous system that may be present in the sthenic form of insomnia. That the first-named of these effects follows its use, I have recently ascertained by experiments upon living animals, the details of which will be given hereafter. Suffice it now to say that I have administered it to dogs whose brains have been exposed to view by trephining the skull, and that I have invariably found it to lessen the quantity of blood circulating within the cranium, and to produce a shrinking of the brain from this cause. Moreover, we have only to observe its effects upon the human subject, to be convinced that this is one of the most important results of its employment. The flushed face, the throbbing of the carotids and temporals, the suffusion of the eyes, the feeling of fullness in the head, all disappear as if by magic under its use. It may be given in doses of from ten to thirty grains, the latter quantity being seldom required, but may be taken with perfect safety in severe cases."

Since then, experiments with the cephalo-hæmometer and ophthalmoscope have abundantly confirmed these views, and more extensive experience in the treatment of cerebral congestion has placed the matter beyond the possibility of a doubt. Other observers have also confirmed the opinions here expressed.

---

[1] "On Sleep and Insomnia." *New York Medical Journal*, June, 1865, p. 203.

The prescription which I usually employ consists of bromide of potassium, ℥ j; water, ℥ iv; of this a teaspoonful is taken three times a day in a little water. Occasionally the bromide is increased to ℥ iss, and sometimes a saturated solution—which contains grs. xxx to ʒ j—is used. I continue the medicine till drowsiness, a slight feeling of weakness in the legs, and contraction of the blood-vessels of the retina—detected by the ophthalmoscope—are produced. The more prominent head-symptoms generally disappear in four or five days, and the results above mentioned ensue in about ten days.

Latterly I have used the bromide of sodium in corresponding doses instead of the bromide of potassium. It is more pleasant to the taste, and does not cause so much constitutional disturbance as sometimes follows the administration of the bromide of potassium in large doses. The bromide of calcium is also well adapted to the treatment of cases of active cerebral congestion, and has the advantage over the other bromides of acting more promptly.

As is well known, ergot possesses the property of constricting the organic muscular fibre. This property has for several years past led to its successful application to the treatment of those diseases of the spinal cord in which it is desirable to lessen the amount of blood in its vessels. It is only lately, however, that this agent has been employed in similar affections of the brain. From my own experience, as well as from a consideration of the investigations of others, I am entirely satisfied that ergot does contract the cerebral vessels, and hence that it diminishes the quantity of intracranial blood. Among the first, if not the very first, to call attention to this property was Dr. Charles Aldridge,[1] who noticed that after the administration of a full dose he found it to cause "contraction of the arteries of the retina and loss of the capillary tint of the disk." My own observations are entirely in accord with these results. I have repeatedly found a single dose of two drachms of the fluid extract produce a decided diminution in the calibre of the retinal arteries, and a marked pallor of the disk.

In addition, some recent experiments which I have performed upon dogs, in which the ergot was administered hypodermically in doses of from one to three drachms of the fluid extract, after the animals had been trephined and the cephalo-hæmometer inserted into the opening in the skull, showed from the falling in the tube that the intracranial pressure was notably lessened.

Applying these facts clinically, it is found that ergot is of very great value in the treatment of active cerebral congestion in all its forms, but especially in the first or hyperæmic stage. I am in the habit of giving drachm-doses of the fluid extract three times a day, in combination with some one of the bromides. An excellent formula is sodii bromidi, ℥ j; ergotæ ext. fluidi, ℥ iv. M. ft. sol. Dose, a teaspoonful three times a day.

---

[1] "West Riding Lunatic Asylum Reports," vol. i., p. 71, London, 1871; also vol. iii., p. 230.

Or the ergot may be given alone, either in the form of the fluid extract, or of the ergotin of Beaujon, which is simply a solid extract. This latter is made into pills of from three to five, or even ten grains each, one of which should be administered three times a day.

In the first or hyperæmic stage, and especially where the pain in the head has been a prominent feature, I have frequently seen prompt relief of the cerebral distress from the administration of ten or fifteen grains of paullinia. The fluid extract, a more eligible preparation than the powder, may be given in doses of a half to a full teaspoonful.

In conjunction with these remedies I very generally employ the oxide of zinc, which experience has taught me is a powerful agent in relieving cerebral congestion, and giving tone to the nervous system. It should be given in doses of grs. ij, three times a day, either in the form of a pill or powder, and to avoid any nausea should be taken after meals.

At the end of about ten days it will generally be found that under this treatment all symptoms of congestion—subjective and objective—have disappeared, leaving a little debility and mental depression. It then becomes expedient to give tonics and restoratives, and those which have a special action on the nervous system are to be preferred. Among them, strychnia, phosphorus, and cod-liver oil stand first.

Strychnia may be advantageously administered in conjunction with iron and quinine dissolved in dilute phosphoric acid, as in the following formula: Strychniæ sul., gr. j; ferri pyrophosphatis, quiniæ sul., āā ʒ j; acid. phosp. dil., zingiberis syrupi, āā ℥ ij. M. ft. mist. Dose, a teaspoonful three times a day in a little water. I prefer this extemporaneous prescription to any of the syrups or elixirs with like ingredients. If for any reason the iron and quinine are not indicated, the strychnia can be given alone with the dilute phosphoric acid.

The eucalyptus, in the form of the fluid extract, has certainly in my hands been productive of excellent results in the treatment of the hyperæmic stage of cerebral congestion. This has been especially the case in those instances in which a malarious influence was present, but in which quinia could not have been given without running the risk of still further adding to the quantity of intracranial blood. An ounce of the bromide of sodium may be dissolved in four ounces of the fluid extract, and a teaspoonful taken three times a day.

Hydrobromic acid is of no service in the treatment of cerebral congestion, except as a solvent for the sulphate of quinia, the injurious effects of which upon the brain it modifies or prevents. A drachm of Fothergill's solution will counteract the congestive tendency of about two grains of the sulphate of quinia. It may in some cases be advantageously substituted for the dilute phosphoric acid of the formula just given.

Phosphorus almost always acts well in such cases as those under consideration. It may be given in the form of the phosphorated oil, as in the following formula: ℞. Olei phosphorat., ℥ ss; mucil. acaciæ, ℥ j; olei bergamii, gtt. xl. M. ft. emulsion. Dose, gtt. xv. three times a day.

A very elegant preparation of phosphorus is the phosphide of zinc. The chemical formula of this substance is $Zn_3 P$, and consequently a grain represents a little more than one-seventh of a grain of phosphorus. The proper dose, therefore, is about the tenth of a grain. I usually prescribe it in cerebral congestion, according to the following prescription: ℞. Zinci phosphidi, grs. iij; rosar. conserv., q. s. M. ft. in pil. no. xxx. Dose, one three times a day. Instead of the conserve of roses, grs. x of the extract of nux vomica may be substituted if strychnia is not being administered in some other form.

Another very useful form for administering phosphorus is the phosphorated resin, which contains four per cent. of phosphorus, thoroughly rubbed up with ninety-six per cent. of resin. This is made into pills with conserve of roses, or some other excipient. The dose is about half a grain, containing the one-fiftieth of a grain of phosphorus.

Latterly I have made much use of arsenious acid in cerebral congestion, especially in cases which have been the result of mental exertion or anxiety. Its action is certainly preferable to that of Fowler's solution. It should be given in doses of about the fiftieth of a grain, and after eating, and should be continued for several weeks. Lisle[1] administers it in the quantity of from a fourth to the third of a grain daily, and there is no doubt that it may be given to this extent without danger. I have never, however, unless there was manifest insanity, used it in these doses.

In those cases in which there are dyspeptic symptoms—and they constitute the majority—the administration of pepsin and powdered charcoal with each meal will be of decided benefit; and in such cases bismuth is often of great service.

Such is the treatment I have found to be most advantageous in active cerebral congestion, and I rarely have occasion to supplement it with other measures, unless some special indication is to be fulfilled. Thus, if the bowels are constipated, a mild purgative may be given, or preferably an enema of warm water or olive-oil; or, if the urine is scanty and high-colored, saline diuretics are useful.

---

[1] "Du traitement de la congestion cérébrale et de la folie avec congestion et hallucinations par l'acide arsénieux." Paris, 1871.

In the passive form of the disease it is sometimes advisable to give stimulants, which may be done from the first in conjunction with the bromide of potassium, sodium, or calcium, with ergot. Alcohol in some form is to be preferred when it is well borne, though carbonate of ammonia is sometimes a useful substitute. In several cases of passive cerebral congestion in old people, and in one notable instance occurring in the person of a very prominent elderly gentleman of this city, I derived the most satisfactory results from sulphuric ether inhaled from a handkerchief to the extent of a teaspoonful, several times a day. The pain, constriction, vertigo, numbness, wakefulness, and inability to exert the mind, were lessened with every dose, and finally entirely disappeared. Ether may likewise be given by the stomach—gtt. xv several times daily—in case the inhalation is contraindicated from any cause.

Of course, any influence capable of interfering with the due return of blood from the head should be counteracted at once.

In the two cases of aphasic cerebral congestion of the passive form, to which reference has been made, I derived the most signal benefit from the use of infusion of digitalis in tablespoonful-doses administered every four hours.

Hygienic treatment should in both types of the disease be persistently carried out. The food should be nutritious, digestible, and ample, though not excessive, in quantity. Alcohol and tobacco, if used habitually by the patient, should be restricted to moderate limits; I have never seen the latter do harm unless used to excess. Tea and coffee may safely be left to the patient's own inclinations and experience. I believe more harm is done by suddenly breaking off a habit, even though it be somewhat injurious, than by tolerating it within due bounds. Exercise in the open air—walking, horseback-riding, or driving—is always beneficial. The same cannot be said of gymnastic contortions, which, to make them worse, are usually performed in hot rooms. Bathing daily and subsequent friction with a tape towel are exceedingly useful in determining blood to the surface of the body. The Turkish bath cannot be too highly commended.

But, above all, those persons who have brought on the disorder by inordinate mental exertion or anxiety, must consent to use their brains in a rational manner if they wish to recover or to avoid future attacks. They have received a warning, and, if they do not heed it, sooner or later other diseases, more difficult if not impossible of cure, will make their appearance.

But it is not always the case that the most positive advice on this point is followed. Men who would readily see the impropriety of walking three or four miles while suffering with an inflamed knee-joint, do not hesitate to exert a disordered brain to the extreme limit of its power. It is impossible that the action of a brain thus affected can be such as to evoke sound and healthy thoughts. It is not to be wondered at, therefore, that the subjects of cerebral congestion who insist upon attending to their avocations, and on concocting schemes for obtaining wealth or fame, should perpetrate acts which result in the loss of fortune, or the acquisition of a reputation far different from that sought.

The cause of cerebral congestion, whatever it be, must, if practicable, be removed, and it must continue removed.

CHAPTER II.

CEREBRAL ANÆMIA.

In cerebral anæmia the quantity of blood in the brain is either reduced below the normal standard, or the quality of the circulating fluid is impoverished. The first-named condition is due either to direct loss of blood, to deficient action of the heart, to impaired nutrition, or to some cause preventing the due access of blood to the brain; the second to disease of some organ concerned in hæmatosis or to a general cachexia.

The two states very often coexist, and they may properly be considered together.

Symptoms.—In cerebral anæmia suddenly induced from profuse hæmorrhage, the most prominent symptom is syncope. Vertigo is generally an attendant, and there are paleness of the features and coldness of the extremities. The pulse is frequent, thread-like, and weak. The respiration is feeble and accelerated.

But, when the accession is more gradual, headache is very generally present. It may be, and usually is, confined to a limited portion of the head, sometimes to a spot not larger than the point of the finger. A feeling of constriction, especially across the brows, is complained of, and the vertigo, notably increased on rising from the recumbent posture, is as troublesome a feature as in the worst attacks of cerebral congestion. There is ringing in the ears, and loud noises are not only painful but are exceedingly irritating to the nervous system. The pupils are largely dilated, and are sluggish, contracting slowly and but little on exposure to a strong light. These phenomena may be restricted to one eye, a circumstance which generally occasions needless alarm on the part of the patient. The retinæ are extremely sensitive, and hence ophthalmoscopic examination is painful. When employed, the vessels at the fundus of the eye are seen to be small and straight, and the choroid is paler than is normal.

Owing to paresis of the ocular muscles—a very common condition in cases of cerebral anæmia—the attempt to use the eyes, as, for instance, in reading, produces pain in them and in the head. In many cases the effort of three or four minutes causes very great uneasiness.

The complexion is pale, the lips almost colorless, or else redder than in health. The skin is cold and clammy.

Nausea and vomiting are present in extreme cases, and convulsions of an epileptic character may occur. In the rapidly-developed form of the disease, caused by sudden and great loss of blood, they are always present, and in the milder and more gradual variety they are occasionally seen. Feebleness of muscular power is always met with, and there may be general or partial paralysis, with the usual derangements of sensibility indicative of anæsthesia, such as coldness, formication, and "pins and needles."

The mind, of course, participates in the general disorder. In extreme cases, due to active hæmorrhage, the patient is completely insensible. In less severe forms there may be all the gradations from low delirium to great mental irritability, or a condition of intellectual lassitude approaching dementia.

Hallucinations and illusions are common in the slowly-developed forms of cerebral anæmia, and may affect any one or all of the senses. Those of sight and hearing are, however, more prominent. In the case of a young lady under my care, and whose only marked disorder was that under consideration, the hallucination that she saw a black man was almost constantly present. At times she conversed with this imaginary being, told him not to trouble her, that she no longer feared him, etc. She believed firmly in his presence, and hence had a delusion.

In all cases of cerebral anæmia there is more or less drowsiness, from the profound syncope of the rapid form to the rather agreeable languor present in slight cases. In instances of medium severity, the patient readily falls asleep in the sitting posture ; but recumbency induces wakefulness, from the fact that the quantity of blood in the brain is thereby suddenly increased above the habitual standard, and a state of comparative hyperæmia is thus induced. I have, in another place,[1] called attention to this form of insomnia, and adduced several cases in illustration.

Examination of the heart by auscultation reveals the existence of bellows-murmurs, both systolic and diastolic. They are heard more loudly at the base of the heart. There are also very generally venous murmurs, which are heard most distinctly in the jugular veins, especially when the head is turned toward the opposite side. Arterial murmurs may also occasionally be perceived.

These sounds are sometimes heard by the patient, and are then exceedingly annoying. I have had under my charge patients suffering from cerebral anæmia, who constantly heard a sound originating apparently in the head, and which, as they described it, resembled that caused by a large shell placed in the ear. That these murmurs are anæmic, is shown by the fact that they disappear under appropriate treatment.

---

[1] "Sleep and its Derangements."

Cerebral anæmia may be of such intensity and be so suddenly developed as to cause almost instant death. Many cases are on record of patients having died with symptoms of apoplexy, and in whom post-mortem examination has shown the blood-vessels of the brain to be empty, and the brain itself pale and exsanguined.

Paralysis of various forms may likewise result from this condition. Sometimes there is hemiplegia, at others paraplegia; again a single muscle or a group of muscles may be affected, and it may even happen that a general state of paralysis may exist. I have frequently seen a single muscle of the eyeball alone involved, and upon one occasion witnessed the loss of muscular power confined to one side of the face in the person of a lady whose brain was evidently very anæmic.

Gintrac[1] cites the following interesting cases communicated to him by Dr. Hirigoyen:

"A young girl twenty years old, affected with amenorrhœa, consulted a midwife, who bled her, attributing her trouble to cerebral plethora. She had hardly lost two hundred grammes of blood when hemiplegia supervened. Iron and tonics entirely dissipated this condition.

"A young woman, twenty-five years old, was subject to a severe epigastric pain, that had been several times relieved by bloodletting. She was thin, pale, and nervous. Nevertheless, a vein was again opened, but only about one hundred and fifty grammes of blood were taken. Notwithstanding this prudence, a syncope ensued while the arm was being tied up, and there were some convulsive movements. After two or three minutes the patient recovered her senses, but was found to be entirely hemiplegic on the left side, and to have some difficulty of speech. Recourse was had to Hoffman's anodyne, valerian, and appropriate food, and at the end of thirty-six hours she was relieved."

A form of cerebral anæmia met with in young children is of great importance, from the fact of its liability to be confounded with another far more dangerous affection, almost its opposite. This was first clearly described by Dr. Gooch,[2] although previously noticed by other observers. In children suffering from this affection, the symptoms, so far as they are noticeable, are similar to those present in the anæmia of adults. The drowsiness is well marked, the head is cool, the pulse is small and weak, the features are pinched, the pupils large and insensible to light, and the fontanelle, if still open, has the scalp covering it depressed. After death, the vessels of the brain are found to be almost empty, and the ventricles distended with fluid. From its resemblance in some respects to hydrocephalus or tubercular meningitis, this affection was called by Dr. Marshall Hall hydrocephaloid. The distinction, however, is so well defined, that none but the most ignorant or superficial observers would fail to recognize it.

---

[1] "Traité théorique et pratique des maladies de l'appareil nerveux." Tome premier, Paris, 1869, p. 548.

[2] "On Some of the most Important Diseases peculiar to Women; with Other Papers." New Sydenham Society Publication. London, 1859, p. 179.

In some cases of cerebral anæmia a tendency to melancholia exists, and positive insanity may eventually result. In most instances of the disease there is mental depression, with a strong predisposition to the production of hypochondria.

Causes.—Hæmorrhage or other exhausting discharge ranks first among the causes of cerebral anæmia. I have known several severe cases induced by epistaxis, and one by the continued loss of blood from leech-bites. Hæmorrhoidal bleeding has also caused it in my experience. No influence of the kind is, however, more common than uterine bleeding, such as occurs before, during, or after labor, from miscarriages and abortions, especially if they are frequently repeated, and from excessive menstrual discharge.

Chronic dysentery and diarrhœa, malarial and other fevers, the rheumatic, strumous, and cancerous diatheses, diseases of the bones and joints, and long-continued purulent discharges, are likewise causes of cerebral anæmia.

I have several times seen the affection apparently caused by congestion of internal organs. Niemeyer, referring to this possibility, cites the fact that it may follow the use of Jounod's boot. At the present time, when this appliance is variously modified and extended beyond its legitimate use by itinerant quacks, it is well to call special attention to this liability. Several cases in point have come under my observation, and in one, a young lady suffering from epilepsy with cerebral anæmia, whom I saw in consultation with my friend Dr. J. Marion Sims, severe paroxysms were induced by each application of the "exhauster." In this case the operator placed the whole body, with the exception of the head, in a vacuum. In another instance, exhaustion from the leg alone caused syncope every time the operation was performed.

Pressure upon or obliteration of the arteries supplying the brain is another cause, and may be produced by ligation of the arteries or by tumors of various kinds. Feebleness of the heart's action, such as results from fatty degeneration, may also occasion cerebral anæmia.

As we have seen, excessive mental exertion is a common cause of cerebral congestion. Strange as it may appear, I have had several cases of cerebral anæmia under my care, in which the disease was clearly the result of a like cause, and these were instances in which the brain had been overtasked to an extreme degree. A little reflection will, I think, show that such cases are strictly in accordance with what takes place in other parts of the body. Thus, we see the moderate use of a muscle or set of muscles increase their size and strength. Inordinate exercise induces hypertrophy, but, if the power of the muscles be still more severely tried, atrophy results. One of the worst cases of progressive muscular atrophy I ever saw occurred in the person of a ballet-dancer, whose gastrocnemii muscles were the apparent starting-points of the disease. Excessive cerebral action produces exhaustion, and exhaustion causes anæmia, as surely as anæmia causes exhaustion.

The action of mental emotions is more obvious. We know that some emotions increase the amount of blood in the brain. Others diminish it, and sometimes with such suddenness as to cause syncope. Fear is one of these, and we have all seen the face become paler under its influence.

Certain medicines are causes of cerebral anæmia, both by their action on the vaso-motor nerves and in diminishing the power of the heart. Tobacco, tartarized antimony, calomel, oxide of zinc, and the bromides of potassium, sodium, calcium, and lithium, are among the chief of these. I was the first to point out this influence of the bromides, and, in a recently-published memoir,[1] have given several cases in illustration of its action. The drowsiness, vertigo, nausea, fainting, weakness of the muscular system, numbness, failure of memory, mental aberration, pallor of the countenance, and anæmia of the retina, all go to show that the quantity of blood in the brain is diminished. Recent investigations not yet published have convinced me that the oxide of zinc acts in a similar manner.

Insufficient nutrition, either from deficient or improper food or disease of the digestive or assimilative organs, is a very common cause. Through its influence not only is the absolute amount of blood lessened, but its quality is deteriorated. The quantity sent to the brain is hence diminished, and that which is supplied is lacking in its proper proportion of red corpuscles. Many of the cases of cerebral anæmia occurring in large cities originate from such influences, and likewise from the vitiated air of narrow and crowded streets, from cold, and from deprivation of light.

Sudden cerebral anæmia may be produced by the shock caused by physical injuries, or even slight surgical operations unattended by effusion of blood. Thus I have several times seen it follow immediately on the passage of a urethral catheter or bougie for the first time.

---

[1] "On Some of the Effects of the Bromide of Potassium when administered in Large Doses." *Quarterly Journal of Psychological Medicine*, January, 1869, p 46. In this paper I stated that one of the most constant phenomena was contraction of the pupils. Very greatly increased experience has convinced me that this is an occasional circumstance, which occurs during the early period of administration only.

The passage of a galvanic current of too great a degree of intensity through the brain may be productive of alarming symptoms due to suddenly-induced cerebral anæmia. Upon one occasion I passed a current from ten cells transversely through the brain of a gentleman—the poles being on the mastoid processes—with the effect of causing syncope, extreme nausea, a cold perspiration on the head and face, and such feeble action of the heart as to cause me to apprehend the most serious results. Placing the head in the dependent position, and causing him to inhale the nitrite of amyl, soon restored him to consciousness, and dissipated the other symptoms.

In another, somewhat similar though not so violent symptoms were induced by the passage of a current from only six cells. Cologne to the nostrils, and a draught of strong whiskey, afforded prompt relief.

These cases, as well as others within my knowledge or experience, show how sensitive some persons are to the primary current, and indicate the care necessary in the use of this powerful agent.

An instance of extreme cerebral anæmia, produced by excitation of the pneumograstic nerve by a galvanic current of too great a degree of intensity, will presently be cited.

Diagnosis.—The principal affection with which cerebral anæmia is liable to be confounded, is cerebral congestion. Indeed, there is no other which can be mistaken for it, if even ordinary perception and judgment be exercised.

From this it may be diagnosticated by the history of the case, and a careful inquiry into the etiology, by the facts that drowsiness, not wakefulness, is a prominent symptom; that the pupils are dilated instead of being contracted; that the pain is more apt to be fixed in a limited part of the head instead of being general; that it and the vertigo are increased by the assumption of the erect position, and diminished by lying down; that the ophthalmoscope shows retinal anæmia; that the face is pale and the skin cold; that the pulse is weak and frequent; and that bellows-murmurs are heard at the base of the heart and in the veins of the neck. The effect of stimulants and tonics in mitigating these symptoms, and the fact that they are increased by exertion and debilitating influences, are also important points to be considered in forming a diagnosis. Attentive consideration of these differential phenomena will prevent a mistake which may be fatal to the patient.

Prognosis.—The prospect of recovery in cases of cerebral anæmia depends mainly upon the removal of the cause, and the adoption of suitable treatment. In those cases which are the result of sudden and profuse loss of blood, the prognosis is grave, and this is especially so if the patient is pulseless and convulsions have occurred. In such instances, even though the hæmorrhage has been arrested, it may be impossible to save the patient.

In the gradually-developed form the prognosis is generally favorable.

Morbid Anatomy.—The vessels of the brain and its membranes are observed upon post-mortem examination to contain less than the normal amount of blood. The tissue of the brain is pale, and section shows a diminished number of the red points in the white substance. Sometimes there is an increased amount of serous effusion in the subarachnoid space, but the ventricles are generally empty.

Pathology.—The questions to be discussed under this head are similar to those connected with the same point in cerebral congestion. That the quantity of blood within the cranium can be diminished as well as increased admits of no doubt, and the fact that the symptoms grouped together as indicating the existence of cerebral anæmia are really the result of deficient blood-supply to the brain is equally certain. The experiments of Kussmaul and Tenner,[1] as well as those of other physiologists, are perfectly convincing.

To observe in man the effects of even temporarily cutting off the supply of blood to the brain, it is only necessary to compress the carotid arteries for a few moments. I have repeatedly done this in rabbits to the extent of producing insensibility and convulsions. Jacobi[2] relates the following symptoms as generally observed in the human subject: Dimness of sight, dizziness, stupor, weakness in the legs, staggering, swooning, loss of consciousness, and sudden apoplectic falling down.

Dr. Alexander Fleming[3] tried the effect of compressing the carotid arteries. "There is felt a sort of humming in the ears, a sense of tingling steals over the body, and in a few seconds complete unconsciousness and insensibility supervene, and continue as long as the pressure is maintained. I have recently performed this experiment several times, with the effect of producing similar phenomena, together with pallor of the countenance, dilatation of the pupils, and temporary headache."

In many cases of cerebral anæmia, the cause, as we have seen, resides in the blood-producing functions, and is such as to cause the formation of blood which does not contain its due supply of red corpuscles. Here, although there may be no diminution in the actual volume of this fluid circulating in the cerebral vessels, the effect is the same so far as the nutrition of the organ is concerned, and hence the symptoms of anæmia are slowly evolved.

---

[1] "Untersuchungen über Ursprung und Wesen der fallsuchtartigen Zuckungen." Frankfurt, 1857. Also, "On the Nature and Origin of Epileptiform Convulsions, caused by Profuse Bleeding," etc. New Sydenham Society Translation, 1859.

[2] Quoted by Kussmaul and Tenner.

[3] *British and Foreign Medico-Chirurgical Review*, April, 1855, p. 529, in a paper entitled "Note on the Induction of Sleep and Anæsthesia by Compression of the Carotids."

Again, it cannot be doubted that spasm of the blood-vessels produced through the sympathetic and vaso-motor nerves explains the origin and continuance of many cases of cerebral anæmia. It is in this way that mental emotions act, and sometimes with such rapidity as to cause instant death. This spasm may be kept up for a very considerable period, with the effect of developing the ordinary symptoms of cerebral anæmia, even after the emotion which originated it has long since disappeared.

Treatment.—The first indication to be fulfilled in the treatment of cerebral anæmia is to get rid of the cause. It often happens that this is still in active operation when patients come under our care, and there is no hope of permanent success till it is removed. Thus, if there is hæmorrhage from a divided vessel, from the uterus, the bowels, the lungs, or other part of the body, it must be arrested; if there is exhausting discharge from the air-passages, the intestines, or the genital organs, it must be stopped; if the digestive or assimilative organs do not perfectly perform their offices, they must be put in good condition; if a tumor or other obstruction to the due course of the blood to the brain exist, it must be removed; and if the hygienic conditions surrounding the patient be bad, or the food inadequate in quantity or quality, they must be improved.

No medicine exercises so powerful an effect in cerebral anæmia as alcohol in some form or other. Perhaps, all things considered, the spirituous liquors, such as whiskey, brandy, and rum, are more generally applicable; for the influence is more rapidly felt, and there is not the same risk of exciting or aggravating gastric disorder as when vinous or malt liquors are used. The quantity must be regulated according to the circumstances of each case, and should always be large enough to materially increase the force of the heart.

But if this were the only effect of alcohol, its benefits in cerebral anæmia would be but temporary, and would certainly be followed by a period of depression. Aside, however, from its stimulating action on the heart, its tendency is to improve the appetite and digestive power, and to relax any spasm of the blood-vessels that may be present.

Occasionally it happens that alcohol is badly borne by anæmic patients. The brain has for so long a time been deprived of a due amount of its natural stimulus—blood—that time is required to enable it to tolerate, and be improved in tone by, the increased supply. Thus the physician will find that in some cases the patients will be apparently rendered worse by the remedy which of all others is calculated to do them most good. The headache and vertigo are increased, the general feeling of debility and *malaise* greatly augmented, and the complaint is made that the liquor has "gone to the head."

Now, it must be recollected that the brains of anæmic persons are in very much the same condition as the eyes of those who have for a long time been shut out from their natural stimulus—light. When the full blaze of day is allowed to fall upon their retinæ, pain is produced, the pupils are contracted, and the lids close involuntarily. The light must be admitted in a diffused form, and gradually, till the eye becomes accustomed to the excitation. So it is with the use of alcohol in some cases of cerebral anæmia. The quantity must be small at first, and it must be administered in a highly-diluted form, though it may be frequently repeated. Cases in which this intolerance of stimulants is exhibited are almost invariably of long duration, and are as those in which from a like cause wakefulness is produced by the recumbent posture.

The carbonate of ammonia, or the aromatic spirits of ammonia, may be given if there are any special reasons why alcohol should not be used, but they are not to be compared to it in efficacy.

In very extreme cases ether is preferable for the time being to any of the foregoing remedies, on account of its diffusive nature; and transfusion may be necessary to save life.

My recent experience disposes me to put a very high value upon the nitrite of amyl in the treatment of cerebral anæmia. Aldridge[1] has shown that it causes, when inhaled, dilatation of the retinal arteries; and the other phenomena of its action, the feeling of fullness in the head and the redness of the face and scalp, unite to prove that it exercises a like effect over the vessels of the brain.

In the cerebral anæmia of weak and chlorotic girls it is especially valuable, although there is no form of the affection, whether transitory or permanent, in which it will not prove beneficial. Even a single dose of four drops inhaled from a handkerchief has repeatedly in my hands relieved anæmic headaches, and effectually dissipated syncope, the result of a feeble action of the heart. Upon one occasion I had, rather imprudently, perhaps, acted in a case of goitrous exophthalmia upon the pneumogastric nerve with a galvanic current of too great a degree of intensity. The heart was rendered exceedingly weak and irregular in its pulsations. The patient, a lady, became insensible from syncope, and was unable to swallow the brandy I held to her lips. I poured a few drops of the nitrite of amyl on a handkerchief and held it to her mouth. Immediately the action of the heart became stronger, the color began to return to the face, and consciousness was at once regained.

In chronic cerebral anæmia, the nitrite of amyl should be administered in doses, by inhalation, of from four to eight drops three times a day. This course may be continued as long as may be necessary, without the slightest deleterious result. I have repeatedly persevered with it for a year, in cases of epilepsy, with the happiest effect. It has never in my experience been requisite to use it longer than a few weeks in cases of cerebral anæmia.

---

[1] "West Riding Lunatic Asylum Reports," vol. i., 1871, p. 77.

Among the more efficacious medicines to be employed in cerebral anæmia, opium and its preparations occupy a high place. Several years since I pointed out[1] the effects of opium upon the cerebral circulation, and, as the result of many experiments, urged that this drug should be used in brain-diseases with due discrimination. It was then shown that small doses of opium increase the supply of arterial blood to the brain. In the treatment of cerebral anæmia I have derived the most decided benefit from doses of opium not exceeding half a grain, and preferably a quarter of a grain, given three or four times a day, and continued for several weeks. An equivalent or even smaller proportional dose of morphia may be exhibited, instead of the entire drug.

It may seem strange, with the cases I have given, and with the knowledge, from experiment and ophthalmoscopic examination, relative to the power of the primary galvanic current applied to the brain or sympathetic nerve to contract the cerebral blood-vessels, that I should recommend the use of galvanism in cases of cerebral anæmia. Clinical experience, however, shows that it is decidedly beneficial, provided the tension be very low. I am satisfied that not more than two or three cells should be brought into action in such cases, and that the current should only be passed for a few seconds at a time.

As adjuncts to these means, the bitter tonics, such as quinine, gentian, columbo, and quassia, are useful. Iron is almost always required, though there are patients who do not tolerate it. In such cases manganese may be substituted with advantage. I have frequently used the sulphate, in doses of five grains, with excellent results. When iron is borne, I know of no better combination than that given on page 68. Cod-liver oil is also a valuable agent in the disease under consideration.

It must not be forgotten that food is the most important factor in relieving chronic cerebral anæmia. The main permanent influence of stimulants and tonics is exerted upon the appetite and digestion, and the blood and tissue forming functions, mainly as an excitant. The real strength must come from the food. This should, therefore, be of good quality; animal food, such as milk, eggs, and meats of various kinds, forming its chief portion.

The influence of position should always be taken advantage of to facilitate the flow of blood to the head, and the erect posture avoided as far as possible, especially during the early stages of the treatment. Thus the patient should be encouraged to pass a good portion of the day in a recumbent position, and should be instructed to assume it at once on the occurrence of any aggravation of the symptoms.

The opposite course is fraught with danger. Physicians are often anxious that their patients should take physical exercise, but it must be remembered that those who suffer from cerebral anæmia have very little vital energy, and a diminished amount of blood is circulating through the organ from which the greater part of their nervous power comes. Muscular exercise lessens the energy, and still further reduces the quantity of blood in the brain, for the muscles require an increased supply while in a state of activity. To be sure, after the strength of the system is in a measure improved, the blood increased in quantity and quality, and the brain supplied with something like its proper proportion, moderate physical exercise is of the greatest service.

---

[1] "Sleep and its Derangements." Philadelphia, 1869, p. 25.

I have several times witnessed severe consequences from the assumption of the sitting or erect position too soon after a profuse hæmorrhage, and in one case death resulted.

As regards mental labor, there is not much need of caution, for the reason that it is impossible for the patient to undertake it to any dangerous extent. But, as he improves in strength, the desire to make use of his increased power may be manifested. It is, therefore, well at this time to prohibit any such exertion as will probably be followed by marked depression. Moderate mental exercise is, however, far from being prejudicial, for it tends to increase the amount of blood in the brain.

Emotional disturbance should also, as a rule, be avoided, although at times it may be productive of great benefit, especially if it be possible to bring into action an emotion contrary to that which may have produced the disease. Thus a lady became subject to cerebral anæmia, directly the result of painful emotions due to domestic trouble. The difficulty was very suddenly removed, or rather the knowledge of its removal was suddenly communicated to her. The reaction was very great; she was thrown into a state of joyous excitement, attended with considerable febrile disturbance, and I was apprehensive for a time that her mind might become permanently deranged, for there were hallucinations and delusions of various kinds, and many symptoms of cerebral congestion. But in the course of a few days, during which she was kept in entire seclusion, and as far as possible from all mental and physical agitation, she entirely recovered both from the secondary and primary disorders.

CHAPTER III.

CEREBRAL HÆMORRHAGE.

UNDER the designation of cerebral hæmorrhage I propose to consider that disease which is often known as apoplexy, hemiplegia, or a paralytic stroke, and which is due to the rupture of a blood-vessel, and the consequent extravasation of blood either into the substance of the brain or into its ventricles.

Two forms of the affection, differing essentially only in the extent or seat of the lesion, but presenting different symptoms, are to be distinguished; these are the *apoplectic* and *paralytic*. In the first there is loss of consciousness; in the second the mind, though perhaps impaired, is not suspended in its action.

Symptoms.—Before the full development of the attack there often is, for several days, a group of symptoms present which indicate cerebral disorder. These are very much of the same character as those denoting the first stage of cerebral congestion, but, though generally not so numerous, are far more striking.

Among the more obvious is a sudden difficulty of speech, arising from slight paralysis of the tongue and other muscles concerned in articulation. Words are not pronounced with the usual distinctness; the tongue seems to occupy more space in the mouth than it should, and is not moved with the requisite degree of promptness and rapidity.

The other muscles on one side of the face may be affected, and hence there is a little distortion, lasting, perhaps, but for a few hours.

Defects of sight may occur, usually characterized by the presence of dark spots in the axis of vision. Such conditions are due to minute extravasations in the retinæ, and are always of most serious importance. I have known retinal clots to precede by more than a year the occurrence of a more severe lesion.

Bleeding from the nose is a common precursor, and, when occurring without being induced by severe muscular exertion, blows, a dependent position of the head, or other obvious cause in a person over the age of forty, is always to be regarded as a symptom of moment.

Numbness limited to one side of the body is of itself sufficient to excite apprehension. I have known several cases in which this symptom was the only premonitory sign. It may be present several days before, or may precede the attack by only a few minutes.

In addition, there may be headache, vertigo, slight confusion of mind, a tendency to stupor, and vomiting.

None of the premonitory symptoms may be present, and then the attack, if of the apoplectic form, occurs with great suddenness. Even if they have been noticed, there is more or less of abruptness in the onset.

Thus the individual is perhaps standing, engaged in conversation, when he is instantaneously struck with unconsciousness, and falls to the ground as if shot; sensibility and the power of motion are abolished, and no signs of vitality are apparent to the ordinary observer, with the exception of the slow and labored action of the heart and respiratory muscles. The breathing is stertorous, the lips and cheeks are puffed out with each expiration, and the pupils are generally largely dilated and insensible to light.

Reflex movements are abolished at first, but after a few moments they reappear, and are even more readily excited than in health, owing to the fact that the controlling influence of the brain is removed.

The voluntary power of swallowing is lost, but it is usually not difficult to cause contraction of the muscles of deglutition by excitation of the pharynx. When these cannot be produced, the prognosis is, if possible, increased in gravity, for the reason that the extravasation is probably in the medulla oblongata, or so situated as to compress it.

The urine and fæces are often evacuated involuntarily.

An apoplectic attack of this character usually terminates in death without the patient recovering his intellect in the slightest degree. If life should be prolonged for thirty-six hours, the probability of a fatal termination is materially lessened. I have never seen a case of cerebral hæmorrhage that was instantaneously fatal, and, although from anatomical and physiological considerations I admit the possibility of such instances, I am persuaded that they must be rare. Jaccoud[1] expresses the opinion that death is immediate in those cases in which the hæmorrhage is in the medulla oblongata, or in those which occur in both hemispheres. Dr. Hughlings Jackson,[2] on the contrary, though conceding from theoretical grounds that hæmorrhage into or near the medulla oblongata might cause instant death, has never witnessed such a termination; and Dr. Wilks[3] says that apoplexy is very rarely, if ever, a suddenly fatal disease, no matter what part of the brain may be the seat of the effusion. Among the reports of several thousand post-mortem examinations at Guy's Hospital, there was but one in which death was asserted to have been instantaneous, and that was a case of meningeal hæmorrhage. Even this was doubtful, for the patient had fallen some distance from the hospital, and was brought in dead.

I have several times had cases under my observation in which, it was said, death had been as sudden as though the individual had been struck by lightning; but careful inquiry and post-mortem examination have either shown that the observers were deceived, or that there had been no extravasation at all, death being the result of heart-disease.

Nevertheless there are instances on record in which hæmorrhage into the medulla oblongata has produced death with as much suddenness as any other possible cause. Ollivier[4] cites a case which came under his observation at the Salpêtrière:

"Batandier (Jeanne Élisabeth), aged sixty-four, of medium height, and inclined to stoutness, was admitted to the Salpêtrière, for attacks of hysteria, with which she had been affected since her seventeenth year, when her menses appeared. These attacks were very violent, and occurred at each menstrual period. They stopped during a single pregnancy at the age of thirty years, and disappeared altogether at forty, when her menses ceased. Her intelligence had not become seriously impaired; she had full power of speech, but complete deafness, existing since infancy, rendered this faculty almost useless to her, and she accordingly communicated with others by means of signs. She was very irascible, her gait was irregular, but nevertheless there was no paralysis. In all other respects her health was good. On the 28th of October, at mid-day, while in the midst of a group of women, she became very angry, uttered a cry, leaned against the wall, and then fell to the ground. She was raised up, but was dead.

---

[1] "Traité de pathologie interne." Paris, 1870. Tome premier, p. 166.

[2] "On Apoplexy and Cerebral Hæmorrhage." "Reynolds's System of Medicine." London, 1868. Vol. ii., p. 520.

[3] "Guy's Hospital Reports," 1866, p. 178.

[4] "Traité des maladies de la moelle épinière." Troisième édition, Paris, 1837, tome ii., p. 140.

"Autopsy forty hours after death. . . . The sinuses of the dura mater were gorged with blood, the pia mater was strongly injected, and easily detached from the cerebral substance ; the middle lobe of the brain presented a well-marked depression ; the brain was firm, and of good consistence; the hemispheres, carefully examined, presented a decided injection of both the white and gray substance, but no hæmorrhagic *foyer*, old or recent ; the ventricles were empty, the choroid plexuses thin and granular ; the optic thalami and corpora striata healthy.

"After having divided the spinal cord below the medulla oblongata, and having removed the medulla oblongata with the cerebellum, and the pons Varolii, a sanguineous clot, irregularly round, and the size of a walnut, was discovered adherent to the posterior part of the medulla oblongata, and extending above as far as the opening into the fourth ventricle, which it entirely closed. The pyramids were not injured, but the olivary bodies were partly destroyed, the right more than the left. The restiform bodies were entirely detached, and were found in fragments in the middle of the clot. The clot was removed and the source of the hæmorrhage was discovered to be in the central gray substance, four or five lines below the inferior border of the pons Varolii, which was a little softer than normal, but which in other respects appeared to be healthy, as did also the cerebellum. An enormous quantity of sanguinolent serous fluid filled the spinal canal, and flowed out in part from the foramen magnum, and in part from the opening made in the spine for the examination of the cord, which was healthy and non-injected.

"Both lungs were gorged with black blood, but presented no traces of emphysema; the right cavities of the heart were filled with black blood, but the organ was healthy.

"All the abdominal organs were in a normal condition."

Ollivier remarks, in reference to this case, that death was as instantaneous as though produced by a sudden luxation of the first or second vertebra.

Dr. A. Charrier[1] has reported the case of a woman who, on the twelfth day after delivery, died instantaneously. At the evening visit, while talking, "she suddenly uttered a cry, turned over on her pillow, and was dead. Death was as instantaneous as though she had been struck by lightning." At the autopsy a small clot was found in the centre of the medulla oblongata. The rest of the brain and the heart were perfectly healthy.

---

[1] "Hémorrhagie du bulbe rachidien." *Archives de physiologie*, 1869, p. 660.

In the majority of cases attended with complete loss of consciousness, the course of the disease is not so rapid or hopeless as in the form just described. The patient falls, is comatose, breathes stertorously, and presents a similar general appearance; but after a time consciousness begins to return, and it is possible to partially rouse him from the condition of insensibility. He turns over in the bed, though with difficulty, and may attempt to speak. Articulation is, however, indistinct, for the muscles of one side of the face are paralyzed, and the tongue, from a like cause, is restricted in its movements. The paralysis is found to exist in the limbs of the same side, and involves the loss of sensibility, as well as of motion, though rarely to the same extent. In some exceedingly rare cases, perhaps not clearly understood, the paralysis of the limbs is on the opposite side to that of the face. A man thus affected was present at my clinic, in October, 1870, at the Bellevue Hospital Medical College. He was a patient under my charge at the New York State Hospital for Diseases of the Nervous System, and had been attacked several years previously. His history, as elicited with great care by my clinical assistant and resident physician of the hospital, Dr. Cross, was perfectly clear on this point.

The facial paralysis presents several points of great interest in a diagnostic point of view. The affected side is incapable of expression, but, so long as the patient does not attempt any facial movements, scarcely any distortion is perceived. Should he endeavor to open his mouth to spit, or to puff out his cheeks, the paralysis is at once noticed. Owing to the fact that the antagonism of the muscles is destroyed, the face is drawn toward the sound side, the angle of the mouth being slightly depressed. It is remarkable, however—and the fact is of importance as a diagnostic mark between the facial paralysis of cerebral hæmorrhage with hemiplegia and the simple facial paralysis from injury or disease of the seventh pair—that the patient does not lose the ability to close the eye of the affected side.

If the fifth pair of nerves is involved in the lesion, sensibility is impaired, which is never the case in simple facial paralysis, and the masseter and pterygoid muscles, which receive their motor influence from this nerve, will consequently be paralyzed. The ability to masticate on the affected side is therefore lost, and the cheek hangs lower than on the sound side.

The tongue is also only paralyzed upon one side. When, therefore, it is protruded from the mouth, the point deviates toward the paralyzed side, owing to the uncompensated action of the sound genio-hyoglossus.

All these paralyses occur on that side of the body opposite to the seat of the lesion. In a very few instances the paralysis has existed on the same side with the lesion. This is explained by the fact that it occasionally happens, as Longet[1] states, that the decussation of the anterior columns of the cord is imperfect. At times, again, owing to a double extravasation, or to the fact that the lesion is in the mesial line of the pons, or that it forces its way so as to involve both hemispheres, both sides of the body are deprived of motion.

Very inexact ideas have prevailed relative to the temperature in cases of cerebral hæmorrhage. The researches of Bourneville[2] have given us more certain data than we previously possessed, and, aside from their value as contributions to symptomatology and pathology, are of great importance in the matter of prognosis. This observer, as the result of numerous determinations, arrived at the following conclusions:

That the animal temperature, in the very inception of the apoplectic attack, undergoes a very considerable reduction, the thermometer in the rectum indicating 36° (=96.8° Fahr.), and even sometimes falling as low as 35.4° (=95.72° F.). This reduction seems to be influenced particularly by the continuance of the hæmorrhage and the supervention of additional centres of extravasation. To this period of temperature-depression succeeds another, during which the animal heat remains stationary at its normal point. If the patient is destined to recover, this period is prolonged indefinitely; but, if death is to ensue, a third period, characterized by a remarkable elevation of temperature, supervenes. During this stage the thermometer indicates 40° (= 104° Fahr.), or may rise to 41.5° (= 106.7° Fahr.).

Charcot[3] has called attention to the fact that, in a few cases of cerebral hæmorrhage, an acute bed-sore forms on the buttock of the paralyzed side. From the second to the fourth day after the occurrence of the attack, an erysipelatous redness of irregular outline occupies the buttock, and frequently extends over the greater part of its surface. Within forty-eight hours a dark-colored spot appears on the central portion, and the epidermis of this is raised by the sanguinolent fluid beneath it. This vesicle breaks, and a sore is thus formed, which gradually extends. Occasionally but very rarely the sore occurs on the sound buttock. I have only witnessed two cases in which these sores were formed, and both were in persons over seventy years of age. Of course, these eschars are not to be confounded with the bed-sores due to long-continued pressure.

It is rarely the case that the third nerve is affected. When it is, there are external strabismus from paralysis of the internal rectus muscle, and ptosis from paralysis of the elevator of the upper eyelid. The pupil is dilated, and is insensible to light.

---

[1] "Anatomie et physiologie du système nerveux," tome i., p. 883.

[2] "Études cliniques et thermométriques sur les maladies du système nerveux." Paris, 1872, p. 116.

[3] "Sur la formation rapide d'une eschare à la fesse du coté paralysé dans l'hémiplégie récente de cause cérébrale." *Archives de physiologie*, 1868, p. 308.

Another phenomenon is sometimes observed, and that is the rotation of both eyes toward the sound side. This is accompanied by a like movement in the head, so that, if the patient is paralyzed on the left side, the eyes and head are turned to the right, and consequently, as the patient lies in bed, the right side of the face rests on the pillow. I have observed these symptoms in about one-third of the cases of cerebral hæmorrhage which have come under my observation. They were present from the very beginning, and disappeared in a few days.

Slight convulsive or involuntary movements are occasionally noticed. The most frequent of these is yawning, a symptom which Dr. Todd[1] regards as troublesome, and even unfavorable, but which, in my experience, is not very annoying or dangerous. The other convulsive actions may be on the whole of either side of the body, or on both sides, or may be restricted to a single limb or even a group of muscles.

Reflex movements are at first sometimes abolished, but subsequently can generally be excited, especially in the lower extremity, by tickling the sole of the foot. Deglutition, though imperfect, can generally be made to take place by reflex action, unless, as previously stated, the hæmorrhage is in, or in the vicinity of, the medulla oblongata.

Strong tonic contractions of the muscles of the paralyzed limbs are occasionally a prominent phenomenon. The upper extremity is more apt to be their seat than the lower, and the biceps and triceps muscles are especially liable to be thus affected. This condition may exist at the very beginning of the seizure, or may subsequently supervene.

Few systematic authors have noticed the symptom in question—a symptom which is not to be confounded with the secondary contractions coming on several weeks after the attack, and the origin of which is altogether different—attention seems to have been first called to it by Boudet,[2] but Durand-Fardel[3] studied it more thoroughly, and was the first to determine its connection with a definite lesion. According to this later author, primary contraction is only present in cases of cerebral hæmorrhage, when the extravasation reaches the ventricles or the subarachnoidal space. So long as the blood remains circumscribed in the cerebral tissue, there are no contractions either in the paralyzed or the non-paralyzed limbs. Of twenty-six cases of cerebral hæmorrhage, in which death ensued within one month, and in which the ventricles or the meninges had been invaded, there had been, in nineteen, contractions of the paralyzed members; in three, contractions of the sound limbs; and in four, resolution without contraction. Charcot,[4] in fourteen cases of ventricular or meningeal invasion, noticed contractions in eleven, and in two epileptiform convulsions. The contractions take place whether the membranes be distended by the clot, or whether rupture ensues.

---

[1] "Clinical Lectures." Second edition. London, 1861, p. 708.

[2] "Mémoire sur l'hémorrhagie des méninges." *Journal des connaissances medico-chirurgicales*, 1839.

[3] "De la contraction dans l'hémorrhagie cérébrale." *Archives générales de médecine*, 1843, tome ii., p. 340. Also "Maladies des vieillards." Paris, 1873, p. 225.

[4] "Nouvelles récherches sur la pathogénie de l'hémorrhagie cérébrale." *Archives de physiologie*, 1868, p. 110.

In the less severe apoplectic form of cerebral hæmorrhage now under consideration, the urine and fæces are sometimes passed involuntarily from paralysis of the sphincters, and are at times obstinately retained from paralysis of the bladder and abdominal muscles.

The mental symptoms are at first scarcely distinguishable from those which are present in the severest form of the disease. The coma and insensibility are complete, but after a time, which varies in duration with the extent of the lesion, consciousness begins to return. The patient opens his eyes, and gives a little attention when loudly spoken to; and is perhaps able to express, to some extent, his wishes by signs and gestures. Gradually the mental power increases; he attempts to speak, but his words are misplaced or forgotten, and his articulation, owing, as already stated, to the paralysis of the face and tongue, is thick and indistinct. Those words which are enunciated by the movements of the lips and tongue are especially troublesome, while those formed in the throat are not difficult to pronounce.

The mental characteristics of the patient will be found to have undergone a radical change. He is irritable, unreasonable, and fretful. His sense of the proprieties of life, which may in health have been very delicate, becomes obtuse; his memory is notably impaired, and his reasoning power greatly diminished. The greatest change, however, is perceived in the emotional faculties. He laughs at the veriest trifles, and sheds tears profusely at the least circumstances calculated to annoy him. Even for years afterward this peculiarity is noticed.

Such is the first stage of an attack of cerebral hæmorrhage marked by apoplexy and paralysis, as ordinarily observed when amendment takes place. It is often the case, however, that this stage is not fully developed, owing to the continuance of the hæmorrhage. In such an event the coma becomes more profound, the breathing more irregular and less frequent, the pulse intermits and loses in force, the face becomes purple from imperfect aëration of the blood, and death ensues. In other cases a certain degree of improvement may be attained, and then the hæmorrhage may recur, and the patient dies comatose.

In a few cases which I have had under my charge, the first symptom observed has been intense pain in some part of the head. This has been quickly followed by nausea and the ejection of the contents of the stomach. There have also been slight wandering of the mind, and a disposition to stagger in walking. These phenomena have persisted for from four to six hours, and then the patients have gradually passed into a comatose condition, with general resolution of the limbs. Death has ensued within twelve hours after the beginning of the symptoms. In one of these cases, that of a gentleman of this city, he had remarked to me, at six o'clock in the evening, that he was feeling remarkably well all day. For several years he had suffered from cerebral hyperæmia, the result of continued and severe mental application. At about eight o'clock he was seized with the most agonizing pain in the head, attended with intense nausea. Repeated vomiting took place, and there had been slight delirium and momentary periods of forgetfulness. My friend Dr. Lente, of Cold Spring, who was in my house at the time, went with me to see him, in response to his message that I would call. We found him as above described; and, as he was firmly convinced that his stomach was at fault, an emetic of salt-water was given him. It acted promptly, but without affording him the least relief. A hypodermic injection of a third of a grain of sulphate of morphia was next administered, but without benefit; and this was followed by a similar quantity after half an hour. He then thought he might sleep a little, but the pain continued. An hour afterward I left him, being of the opinion, in which Dr. Lente shared, that he was either suffering from a cerebral tumor or an extravasation of blood. Two hours afterward I was again sent for. He was then comatose, the limbs in a state of resolution, the breathing of that loud, rauchous character, and the heart beating with the irregularity so indicative of effusion into, or in the neighborhood of, the medulla oblongata. Deglutition could not be excited by substances placed in the mouth. The right pupil was strongly dilated, while the left was a mere point. Death ensued within two hours afterward.

The post-mortem examination was made the next day by Dr. S. D. Powell, in presence of Drs. Lente, Ripley, Elsberg, and myself. A clot the size of a small orange occupied the posterior part of the middle and central portion of the right lobes. It was entirely confined to the white substance. Another, about as large as a hickory-nut, was situated in the right half of the pons Varolii.

In all probability the clot in the right hemisphere began to form first, and that the second, into the pons Varolii, which was the immediate cause of death, did not originate till a considerably later period, indicated by the disturbances in the respiration and circulation, and the impossibility of exciting deglutition.

In those cases in which the improvement has been progressive up to the point of partial resumption of the mental faculties, we find that a second stage characterized by different symptoms often supervenes. This is the period of inflammation.

It may begin at a variable time after the occurrence of the extravasation, usually not later than the eighth day. It is marked by febrile excitement and pain in the head, the latter being often very severe. There is gastric derangement, as evidenced by nausea and vomiting; and convulsive movements of the limbs, with contractions of the flexors of the paralyzed side, are generally present. Delirium is also a prominent feature. Sometimes there is obstinate wakefulness, and at others a strong tendency to coma. This stage may last three or four days, or at most five or six, when it either causes death by extension of the inflammation from the immediate vicinity of the lesion to other parts of the brain, terminates in the formation of an abscess, or gradually ends in resolution, with abatement of the symptoms.

Disregarding for the present the first two of these results, we proceed with the consideration of the phenomena of a case in which resolution takes place.

With the cessation of the inflammatory action, the improvement of the patient becomes very marked. His speech is every day more distinct, his mind more active, his paralyzed limbs more capable of motion. Usually the leg recovers power with much greater rapidity than the arm, and thus the patient is able to walk tolerably well before he can raise his arm from his side, bend the elbow, or extend the fingers. The paralysis in the leg is most marked in those muscles whose office it is to elevate the foot, and this necessitates a peculiar gait in order to avoid dragging the toes along the ground. The abductors are rarely affected to any great extent. The patient in walking, therefore, throws the leg out from the body, and then, swinging it around, clears the ground in this manner.

In the upper extremity there is almost invariably a disposition toward contraction of the pectoralis major and minor muscles, by which the arm is drawn across the front of the thorax. At the same time the latissimus dorsi, the trapezius, the rhomboidei, the teres major and minor, are generally in a state of relaxation, and eventually tend to atrophy. The elbow is slightly flexed, the wrist bent upon the forearm, and the fingers drawn in toward the palm of the hand. These actions may, in a great measure, be prevented by appropriate treatment, and they may vary in extent according to the gravity of the attack. It is a curious fact that the muscles of respiration are never paralyzed in cerebral hæmorrhage unless the medulla oblongata be involved.

Trousseau[1] has insisted, with great force, on the fact that, when the arm regains power before the leg, the termination is always fatal. That this is the general result, I am very sure from my own experience, but it is not invariable, for there are now in the New York State Hospital for Diseases of the Nervous System two patients affected with cerebral hæmorrhage whose arms have improved to a very great extent, while the legs are still as much paralyzed as ever.

Now, with all these troubles of motility, sensibility may likewise be involved to a greater or less extent. When this is the case, the limbs of the affected side at first feel heavy as if made of lead, and after a while numbness, as exhibited by a feeling as if ants were crawling over the skin, or water trickling over it, as if pins and needles were sticking in it, or as if that part of the body were "asleep," is noticed. Sometimes the sense of touch is greatly lessened, while the ability to feel pain is scarcely impaired, and indeed is often considerably increased. Again, there may be hyperæsthesia of the skin of the affected regions, and pain along the course of the nerves.

---

[1] "Lectures on Clinical Medicine." Bazire's Translation. Part I. London, 1866, p. 16.

The circulation is inactive in the paralyzed limbs, and this, together with the deficient nervous power, tends to cause a permanent reduction of temperature. The difference may amount to as much as five or six degrees, and, as the ability to resist cold is diminished, the patient is obliged to use additional covering on the paralyzed members.

From continued disuse, atrophy of the paralyzed muscles always takes place unless suitable treatment be begun at an early period.

Thus far we have only considered those attacks of cerebral hæmorrhage which are accompianed with unconsciousness. One of these forms kills, without the patient so far recovering as to show whether he is paralyzed or not, though of course he is so to a profound degree; the other allows of more delay; the brain can still act to some extent, and, if death does not ensue from continuance of the hæmorrhage, the patient is found to be paralyzed on the side of the body opposite to the seat of the brain-lesion. One other form requires notice, and it is, perhaps, the one most frequently met with. It differs from the attacks just described, in the important fact that it is unattended with unconsciousness.

Like the others, this species of cerebral hæmorrhage may take place very suddenly, without premonitory symptoms, or it may, like them, happen while the patient is said to be asleep. Generally, however, though there may be no long prodromatic stage, there are symptoms occurring immediately before the attack which indicate both mental and physical disturbance. These are headache, vertigo, numbness, vomiting, irritability of temper, and, perhaps, slight difficulties of speech.

When the attack comes, the individual, if standing, falls, from the immediate paralysis of one leg. He is fully sensible of his condition, although there is generally more or less mental change. The arm and face are affected, and the speech is rendered impossible or indistinct.

If the patient be sitting or lying, he is aware that something has happened, but does not discover its exact character till he attempts to rise. A distinguished general officer of the army, after a fatiguing day of ceremony, entered his carriage with his wife, to be driven to his hotel. As he passed along Fifth Avenue, he felt an indescribable sensation, and immediately afterward noticed that he could only see the half of objects. He made no effort to speak, though he is confident he did not for a moment lose his consciousness. When he attempted to get out of the carriage, he found, to his surprise, that he was paralyzed on the right side, and that his speech was so much impaired that he could not be understood.

Another gentleman was reading an amusing book, at which he laughed heartily. He felt suddenly a feeling of vertigo, and the book dropped from his hand. He attempted to pick it up, but found he had lost power in the arm, and, on trying to call to his wife, who was in the same room, discovered that he could not speak. At this time he could walk, but in a moment or two afterward he fell, from paralysis of his leg. So far as the paralysis is concerned, I have rarely seen a more severe case than this.

Another went to bed, perfectly well, to all appearance, having enjoyed uninterrupted good health for several years. In the morning he arose, but felt a little pain in his head. As he stood before his glass, he thought his face was slightly twisted, and he noticed as he was shaving himself that he did not feel the razor on one side. While he was testing his facial mobility and sensibility, he experienced a trace of numbness in his left hand. This gradually increased, and in addition the limb lost power. In a few minutes he could not move it at all. By the time I saw him—two hours afterward—the paralysis had extended to the leg. At no period was there insensibility or mental confusion.

A gentleman retired at night in good health. On attempting to get out of bed he discovered that he was paralyzed in the leg. Neither the arm nor the face was affected.

In the case of a gentleman of this city whom I saw in consultation with Dr. W. M. Polk, and who had for several years suffered from frequent severe headaches and other cerebral symptoms, the only phenomenon was binocular hemiopia, with occasional slight delirium. Dr. H. Knapp, who saw the patient before I did, discovered no alterations in the functions or structure of the eye, and we all agreed that the case was one of very slight cerebral hæmorrhage.

Several cases have been under my care in which only the face or the tongue was paralyzed; others in which the arm alone was involved; and others, like the one just mentioned, in which the symptoms were confined entirely to the leg. Sometimes there was a momentary feeling of vertigo, sometimes a vacant stare, something like that of the *petit mal* of epilepsy, sometimes a slight degree of intellectual confusion, sometimes headache, and, again, no head-symptoms whatever. The subsequent progress of such attacks requires no special consideration beyond that already given to the more severe forms.

Now, no matter how light the attack may have been, nor how rapid the improvement, the patient who has had cerebral hæmorrhage is never mentally or physically the same as he was before. If the seizure has not been severe, he may advance so far toward a complete cure as to evince very little disorder of his mind or body. But close observation shows that he is not entirely restored, and, though he may do very well for light intellectual and physical exertion, severe labor of either kind is beyond his powers—and no one is more sensible of this fact than himself. Ev after years his emotions are abnormally excitable. A patient now in the New York State Hospital for Diseases of the Nervous System informs me that he sheds tears every time a funeral passes him, and that even hearing of any one's death, or reading the obituary column in a newspaper, causes his feelings to get the better of him. In the lightest forms of the attack, this easily-aroused emotional disturbance is a marked feature for years subsequently, if it ever entirely disappears. And as regards the muscles which have been paralyzed, it is very certain that, though they may be made strong enough for all practical purposes, they never can be restored to their former sound condition.

The character and general mental type of the individual usually undergo some change; and this may be to the extent of reversing his ordinary traits.

Causes.—Advanced age is one of the most influential circumstances which predispose to an attack of cerebral hemorrhage, and this fact has long been known. Thus Hippocrates[1] states that apoplexy is most common between the ages of forty and sixty, and modern investigation establishes the truth of the proposition as regards the actual number of cases. It is probable, however, that the liability increases, as Dr. Flint[2] says, from the age of twenty upward, and that there are not so many cases occurring in persons over sixty as below, for the reason that the number of individuals alive of that age is less.

Of three hundred and eighty-three cases of cerebral hæmorrhage which have been under my professional care, at some time or other after the occurrence of the extravasation, in my private and hospital practice, and in which the age of the patient is noted, three hundred and forty-one occurred in persons over forty years of age. Of these, three hundred and eleven were between forty and sixty, thirty-three between sixty and seventy, five between seventy and eighty, and three over eighty.

Of the thirty-one cases in persons under forty, twenty were between forty and thirty, ten between thirty and twenty, and one under twenty. This latter was a boy of seventeen, whom I exhibited at my clinic at the Bellevue Hospital Medical College in the autumn of 1870.

The disease is certainly more common among men than women, though some authors have asserted the contrary. Falret ascertained that, of twenty-two hundred and ninety-seven cases, sixteen hundred and sixty occurred in males and only six hundred and thirty-seven in females. In my own experience, of three hundred and eighty-three cases, two hundred and fifty-nine were in males and one hundred and twenty-four in females.

Temperament and organization are supposed to have an influence in predisposing to cerebral hæmorrhage. It was formerly thought that those of sanguine temperament and plethoric habit who had stout bodies, large heads, florid complexions, and short, thick necks, were especially liable; but more exact and thorough investigation would appear to show that such is not the case, and that thin and pale individuals show fully as great a proclivity. Dr. Flint[1] expresses the opinion that there is no special apoplectic constitution, and my own experience is decidedly to the same effect.

---

[1] "Aphorisms," chapter vi., aphorism 57.

[2] "A Treatise on the Principles and Practice of Medicine." Third edition, Philadelphia, 1868, p. 582.

That the tendency to cerebral hæmorrhage is often hereditary, appears to be very certainly established. Within my own knowledge, I am aware of several striking instances which support this opinion. A gentleman consulted me for hemiplegia, the result of cerebral hæmorrhage, whose grandfather, father, two uncles, two brothers, and one sister, had died of this disease, and whose son, thirty-six years of age, had been attacked. In another case a lady had her father, two brothers, and one sister, die of the disease; and, in a third very remarkable case, the great-grandfather, grandmother, father, four uncles and aunts, and two brothers, all in a direct line, died of cerebral hæmorrhage.

Piorry[2] cites the case of a woman, herself paralytic, whose three children had died of convulsions, and whose mother, uncle, and brothers and sisters, to the number of twelve, had died of cerebral hæmorrhage or convulsions. It has very often happened in my experience that the father or mother of a hemiplegic patient, whose condition resulted from cerebral hæmorrhage, had been affected in a similar manner.

As regards the influence of diseases of the heart, Legallois, Bricheteau, Rostan, Andral, and Bouillaud,[3] adduce instances in support of the existence of a definite relation. While others, among whom Rochoux, Walshe, and Flint, are to be placed, deny the existence of any such causative influence. As tending to produce active or passive cerebral congestion, disease of the left or right side of the heart would reasonably seem to be conducive to the occurrence of cerebral hæmorrhage. The tension of the blood in the vessels of the brain is increased thereby, and the liability to the rupture of a diseased vessel rendered greater.

The condition of life has also been supposed to exert an effect in predisposing to cerebral hæmorrhage, it being asserted by some authors that the affection is much more common with the rich, and those living in ease, luxury, and refinement, than in the poor and laboring classes.

It is difficult to arrive at any very definite conclusion on this point, owing to very obvious reasons, but I am inclined to think the theory to be not well founded. It is only necessary to visit our large hospitals, to see how many of the inmates, drawn as they generally are from the laboring classes, are suffering from cerebral hæmorrhage or its effects.

Thus far we have only considered the more important, intrinsic,—

---

[1] *Op. cit.*, p. 583.

[2] "De l'hérédité dans les maladies," p. 107.

[3] "Traité de clinique des maladies du cœur," second edition, tome ii., p. 580.

predisposing causes; there are, however, others which may be called extrinsic.

Season is one of the chief of these. The disease is much more common in winter than in the other seasons, although some statistics would seem to show more cases during summer. A careful examination of such, however, shows that under the head of apoplexy is included not only cerebral hæmorrhage, but congestion, sunstroke, embolus, and in fact nearly every other affection attended with sudden loss of consciousness. My own researches have been very exact on this point, and as their results I find that, of the three hundred and eighty-three cases of which I have notes, one hundred and forty cases occurred in winter, eighty-one in spring, ninety-seven in summer, and fifty-eight in autumn. It has been noticed, too, that sudden variations of temperature, especially from mild to cold weather, increase the number of cases of cerebral hæmorrhage.

Of the exciting causes, a long list can readily be made. Among them are the excessive use of alcoholic liquors and other stimulating substances; the use of opium in excess; the ingestion of large quantities of food, especially such as is stimulating and indigestible; excessive physical or mental exertion, strong emotional disturbance, such as anxiety, extreme joy, anger, or terror; the act of coition, especially in old people; straining at stool; enlarged prostate, or paralysis of the bladder, requiring strong muscular efforts for the evacuation of the urine; childbirth; tight clothing about the neck, chest, or abdomen; certain occupations which require the head to be depressed; vomiting, sneezing, coughing, and laughing; exposure to the direct rays of the sun or other sources of great heat; the sudden arrest of a customary flux, such as hæmorrhoidal bleeding; the sudden application of cold water to the body; long-continued bathing in very warm water; the circumstance that the patient has had a previous attack, and certain diseases, as gout and syphilis.

In regard to some of these causes, I may state that several very interesting cases have occurred in my own practice. In one, a lady was attacked on hearing that her cook had left her; in another the emotion excited by the fall of a picture from the wall caused a seizure. Four cases produced by straining at stool have come under my observation. In one of them a gentleman well known in public life retained sufficient consciousness and intelligence to take a large key out of his pocket with the non-paralyzed hand, and to rap on the floor for assistance.

Two cases occurred during sexual intercourse, one in a man, the other in a woman. In one of these there was, subsequently, a great increase of venereal desire. In one case, the seizure was induced by stooping over to tie the shoe. This was in the boy, seventeen years of age, already mentioned. It must be confessed, however, that very frequently, perhaps in the majority of cases, no immediate cause can be reasonably alleged. Of the three hundred and eighty-three cases noted by myself, no cause was noted in two hundred and ten.

Relative to the influence of sleep, I am by no means in accord with those authors who regard it as a powerful exciting cause. During sleep the quantity of blood circulating in the cerebral blood-vessels is diminished, and hence there is less tension upon their walls than during wakefulness. I doubt very much whether cerebral hæmorrhage ever occurs during healthy, undisturbed sleep.

But there is a condition which supervenes upon sleep, and which, to ordinary observers, presents the usual phenomena of sleep, but which is really a very different state, both as regards the brain and the symptoms—and that is stupor due to venous congestion. In this affection there is an increase of the pressure upon the brain, produced by the overdistended vessels; and hence coma, to some extent, ensues. This state is characterized by difficulty of awaking the individual, by turgescence of the larger veins of the neck, by a more or less purple hue of the face, by snoring, and by the puffing out of the lips and cheeks in breathing. Both of these latter phenomena are due to paralysis.

In this condition it is not unusual for cerebral hæmorrhage to occur, but the existing state is not sleep.

So far as my own experience extends, I have not found a majority of the cases, where I have examined into this point, to have taken place either during sleep or the stupor to which I have referred. I have made it a rule, not only in those cases of cerebral hæmorrhage which have been under my own care, but all others, in which I could do so, to inquire particularly with reference to the matter in question, and have found that, in three hundred and eighty-five out of four hundred and sixty-seven cases, the individuals were awake at the time of the attack.

Doubtless much of the confusion has arisen, not only from the non-discrimination of sleep from stupor, but also from treating of apoplexy as a disease instead of regarding it as a symptom due to several very different pathological conditions of which cerebral hæmorrhage is only one, and of which embolism, thrombosis, congestion, meningeal hæmorrhage, and epilepsy, are others.

Finally, it may be said of the etiology, that whatever tends to increase the flow of blood to the head, or to retard its exit, is capable of acting as an immediate cause of cerebral hæmorrhage.

Diagnosis.—The diagnosis of cerebral hæmorrhage is ordinarily not difficult, but it must be confessed that one or two affections are very liable to be confounded with it, and the attendant circumstances surrounding a patient in a condition of insensibility may be such as to materially increase the obstacles to the formation of a correct opinion.

Thus, supposing an individual to be found in a state of profound insensibility, the condition may be due to compression from injury of the skull, to concussion from a fall or blow, to congestion, to asphyxia, to syncope, to a recent epileptic fit, to uræmic intoxication, to hysteria, to narcotism, or to drunkenness.

A mistake of either of these states for cerebral hæmorrhage would be, in the end, embarrassing to the physician, and perhaps injurious to the patient.

The coma might also be the result of embolism, of thrombosis, of tumor, of abscess, or of meningeal hæmorrhage ; but, as regards these conditions, no opprobrium could be attached to the physician, or harm come to the patient, by any error of diagnosis, although a regard for scientific exactness should always prompt us to be as specific as possible in our inquiries and examinations.

From asphyxia, cerebral hæmorrhage is distinguished by the fact that in the former the respiration is suspended. The cause is often apparent. A careful examination of the cranium, and a survey of the surrounding circumstances, will enable the physician to ascertain the existence or non-existence of compression from traumatic cause. This cause may either be depression of bone, the rupture of an internal blood-vessel, or the entrance of some foreign body, as a bullet, into the interior of the skull. So far as symptoms are concerned, there might be considerable difficulty in diagnosticating either of these accidents from cerebral hæmorrhage, but the history would render a mistake impossible.

Concussion presents more difficulties, because the comatose person may be found in such a situation as to warrant the opinion that he has fallen from a height, or otherwise received a blow on the head, when in fact he is suffering from cerebral hæmorrhage. But if he has fallen from a height or been struck, there will probably be more severe bruises about his person than if he is affected with cerebral hæmorrhage, and there may be bleeding from the ears or nose—symptoms of cranial injury not met with in the latter condition.

If, however, the individual has fallen from a height, he may have done so in consequence of an extravasation of blood in his brain, and he may present all the marks of suffering simply from the concussion, or he may have fractured skull with compression. It is, therefore, impossible to make a correct diagnosis in all cases, or to lay down any certain rules which will constitute infallible guides. It is perfectly possible to meet with cases such as those referred to, in regard to which no human judgment can be certainly correct. Such instances are of course rare, and accordingly, in the great majority, the circumstances and the presumption will generally lead to a correct opinion.

From congestion of the apoplectiform variety cerebral hæmorrhage can generally be distinguished without much difficulty. The absence of stertorous breathing, the short duration of the coma, the transient character of the paralysis, the contraction of the pupils, the fact that the loss of sensibility and the power of motion are not generally confined to one side of the body, and the longer continuance of premonitory symptoms, will be sufficient indications of the existence of congestion. Syncope is distinguished by the circumstances that the respiration and circulation are both diminished in power if not suspended, that there is no hemiplegia, that the face is pale, the skin cold, and that these phenomena are all transitory in character. The history of the case will also assist us in arriving at a correct judgment.

Epilepsy, if seen from the beginning of the paroxysm, cannot be mistaken for cerebral hæmorrhage, nor this latter for epilepsy, if the onset of the attack has been witnessed. Even if there are convulsions present in the apoplectic seizure, the error could not readily be committed if attention be paid to the attendant phenomena. For there is no biting of the tongue, the convulsions are persistent, and the animal heat is lowered, whereas in epilepsy the temperature rises at once and remains high—105° Fahr. or thereabouts, during the convulsive stage. But the person found in a comatose condition, with no previous history to guide us, may be supposed to be either in the comatose stage of an epileptic paroxysm, or to be laboring under a seizure due to extravasation of blood. In such a case, if the fit has been epileptic, foam will be found around the mouth, and perhaps blood from injury of the tongue or cheek. Moreover, the stupor of epilepsy is not usually of long duration, and is not generally characterized by stertorous breathing.

In uræmia, the coma of which is very similar to that resulting from cerebral hæmorrhage, the history of the case is our chief reliance for a correct diagnosis, though the absence of hemiplegia and the general presence of anasarca are of course of great value. Moreover, in very doubtful cases the urine may be drawn off by the catheter, and examined for albumen and tube-casts. If these are present, the probability of the stupor being due to Bright's disease and uræmic intoxication is very much increased. The fact, also, that in uræmia there is a progressive fall of the animal temperature—as low as 91.5° Fahr. being reached — and that there is no subsequent elevation, are important points in this connection.

Coma is sometimes a manifestation of hysteria, but a very little acquaintance with the phenomena of this condition will suffice to prevent mistakes. In some cases of hysterical coma there is well-marked hemiplegia; but even when this complication is present, the facts that the hysterical diathesis exists, that there have probably been other manifestations of hysteria, that the pulse is small, weak, and frequent, and that the breathing is free from stertor, will enable a correct diagnosis to be formed.

In narcotism the condition often bears a close resemblance to that due to cerebral hæmorrhage. But in the former there is no hemiplegia, the pupils are generally contracted, the respiration is not stertorous, and the coma comes on gradually.

Drunkenness and cerebral hæmorrhage are often confounded. I have known some sad mistakes of the kind to be made, both by professional and non-professional persons, many of which were unavoidable, for it must be confessed that there are great difficulties connected with the subject. The habit of drinking alcoholic liquors is so general that no reliance can be placed upon the test of smelling the breath. A person may have just taken a glass of wine or of brandy, and be seized with extravasation of blood in his brain immediately afterward, and when not in the least intoxicated. And, even if dead-drunk, he may at the same time have cerebral hæmorrhage. In such a case as the latter, discrimination would be impossible. In ordinary cases of alcoholic intoxication the patient can generally be roused to some extent; the pupils are dilated, but this latter is often the case in hæmorrhage; the breathing is usually free from stertor, but some drunkards always snore; the pulse is small and weak, and there is no hemiplegia. When all these symptoms are in accord, there will be little difficulty; when they are not, the physician must be guarded in his expressions of opinion, and diligently inquire into the personal characteristics of the patient and all matters bearing on the history of the case.

From the centric diseases previously mentioned, the diagnosis of cerebral hæmorrhage is easy as regards some, and difficult as to others. Thus, from embolism it cannot in many cases be distinguished in the first stage. But when all the phenomena are taken into consideration the chance of error is very much diminished. Embolism is generally accompanied with disease of the left side of the heart, and there is often a history of rheumatism; there are never any premonitory head-symptoms; it occurs in young persons as well as old; for reasons which will be explained when the subject of partial cerebral anæmia from embolism is considered, the resulting hemiplegia is generally on the right side; the paralysis generally disappears in a few hours after the attack; if it does not, there is no gradual improvement, as in cerebral hæmorrhage; there are no contractions or partial convulsions,[1] and there is more frequently delirium.

The gradual development of the symptoms in thrombosis, tumor or abscess, and the frequency with which convulsions ensue in the latter diseases, together with the associated symptoms, will prevent the coma which sometimes exists being mistaken for the stupor of cerebral hæmorrhage.

During the subsequent stages of cerebral hæmorrhage, when the mental condition and the hemiplegia are the most prominent features, inquiry into the antecedent history will bring out the foregoing points, and assist us in arriving at a correct idea of the cause. Even, however,

---

[1] Jaccoud (*op. cit.*, p. 141) so asserts, though I have seen one case in which post-mortem examination revealed the presence of an embolus in the middle cerebral artery, and in which there had been convulsions.

should we be baffled in this respect, no great inconvenience could res
either to the patient or physician.

Prognosis.—The prognosis depends upon the extent or situation
the hæmorrhage, and refers to the probability of saving life during
period of attack and immediately afterward, and of curing or mitigati
the subsequent paralysis.

In the severe apoplectic form, death is almost inevitable; so far
my experience goes, it is the invariable result. It generally takes pl
within a few hours. If, however, life be prolonged till the fourth d
there is some hope. Irregularity of pulse, or one very rapid, impo
bility of swallowing, involuntary evacuation of the fæces, and c
sweats, render, if possible, the prognosis still more unfavorable.

In the apoplectic form attended with paralysis, the gradual incre
of the coma and hemiplegia indicate the continuance of the hæm
rhage, and are consequently of grave importance. About one-third
those attacked with this form die. The prognosis is bad in proport
to the debility and age of the patient, and the circumstances un
which the attack has occurred. Thus, if it has supervened in a pers
who has had no obvious exciting cause, the probability is that the
serious disease of the blood-vessels, whereas, coming on in a young p
son as the result of severe muscular exercise, or mental strain, the pr
nosis is more favorable. A second attack is more apt to prove fa
than a first, and a third than a second, and so on.

In the mild form characterized by paralysis, but no loss of conscio
ness, the prognosis is generally favorable. It must be recollected, h
ever, that the risk of inflammation is quite great, both in this and
apoplectic form with paralysis, and that the patient is not safe from
till after the eighth day.

And in both forms, if the temperature rise above 100° Fahr.; if
respiration be chiefly abdominal; if the patient is unable to swall
and rattling of mucus is heard in the throat, the prospect of recover
bad. The same may be said of pain in the head and contractions of
paralyzed muscles. If, further, as Bourneville has shown, the tempe
ture reaches 104° Fahr., death is inevitable.

As regards the probability of recovery from the paralysis, much
pends upon the opportunities the patient may have for receiving pro
medical treatment. The tendency is generally toward amendment e
in the worst cases. Gradually the speech improves, the breathing
comes better, and the arm acquires more strength; but the impro
ment often stops here, and never goes on unaided to complete recove
The longer the paralysis has lasted, the less prospect there is of gr
progress under any treatment; and, if strong contractions produci
distortions have taken place, the prognosis is unfavorable.

Certain muscles recover better than others. The extensors of t foot and hand are especially intractable, but, as a rule, those of the lower extremity improve more rapidly than those of the upper.

The mind ordinarily improves, *pari passu* with the physical symptoms, though not always. I have witnessed several exceptions to the rule. Even in slight cases the intellect may suffer to a great extent, and in no case is it ever in all respects as good as before the attack. Among the unfavorable signs are, persistent irritability of temper, failure of memory, and the existence of delusions. Difficulties of speech, whether as regards the memory of words, or the ability to coördinate the muscles of speech, so as to pronounce them properly, are often very persistent. I have now under my care a gentleman who was attacked with cerebral hæmorrhage two years ago, whose physical powers are quite good, and whose mind is not seriously impaired, but who cannot yet remember sufficient words to carry on an ordinary conversation. When the difficulty is simply due to paralysis of the tongue and facial muscles, the prognosis is more favorable.

Morbid Anatomy.—The seat of the extravasation from cerebral hæmorrhage may be in the substance of the cerebral tissue, or in the ventricles. The former is much the more common.

Now, the blood, which is poured out from a ruptured vessel into the substance of the brain must, of course, occupy its place by separating or lacerating the fibres. It thus forms for itself a cavity, which enlarges as the hæmorrhage goes on, until at last the resistance to further separation or laceration may be so great as to overcome the tension of the blood, and thus put a stop to the bleeding.

The shape of the cavity varies according to the manner by which it has been produced. When it is formed by the separation of the cerebral fibres, it is generally elongated; whereas, when produced by laceration, it is oval, round, or irregular in form. The situation of the hæmorrhage modifies the form of the cavity. In the hemisphere it is usually round; in the motor tract, irregular or oval. The variations as regards size are great. I have seen clots no larger than a pea, and again as large as an orange. When hæmorrhage occurs in the motor tract, the clot is almost invariably small; whereas, in the hemispheres, in the cerebellum, or in the ventricles, it is large.

A clot does not always consist of blood alone. Brain-tissue is very often mixed with it, and this is especially the case when the extravasation has been into the white substance of the hemispheres.

Gintrac[1] has collected the data of five hundred and sixty cases of cerebral hæmorrhage, in which there was a single clot, and in these the seat of the extravasation is shown in the following table :

---

[1] "Traité théorique et pratique des maladies de l'appareil nerveux" Tome deuxième. Paris, 1869. Art. "Hémorrhagies du cerveau."

Corpora striata	72
Optic thalami	38
Corpora striata and optic thalami simultaneously	48
Middle lobes of the brain	127
Pons Varolii and crura cerebri	78
Cerebellum	36
Ventricles	46
Posterior lobes of the brain	33
Anterior lobes of the brain	17
Medulla oblongata	2
Corpus callosum	1
Cortical substance of the brain	45
Total	560

The ordinary seat of cerebral hæmorrhage is thus seen to be the motor tract, for in nearly one-half of the total number of cases the lesion was situated either in the corpora striata, the optic thalami, the pons Varolii, the crura cerebri, or the medulla oblongata. And of these parts the corpora striata and optic thalami are preëminently liable. Next in order of frequency come the middle lobes.

In the great majority of the cases of cerebral hæmorrhage the lesion is situated primarily in the gray substance. This is probably due to the fact of the greater vascularity which this tissue possesses. It would appear, too, that even when the extravasation is not into the corpus striatum or optic thalamus, it is very apt to be in the immediate vicinity of these organs. M. Duret[1] has given an anatomical explanation of this fact, which appears to be satisfactory. According to this observer, the arteries of the corpus striatum, which are given off generally from the middle cerebral artery, though sometimes from the anterior cerebral, enter the brain through the anterior perforated space. A few delicate branches go to the ventricular ganglion of the corpus striatum, but the larger ramifications are distributed sometimes to the external nucleus of the corpus striatum, but more generally they wind around this organ, and give origin to branches which are widely distributed, reaching even as far as the island of Reil. Thus the largest intra-cerebral arteries are situated in the external portion of the corpus striatum. And this is the exact place where, according to Charcot, cerebral hæmorrhage is most apt to occur.

Gendrin[2] had previously remarked that the extravasation in cases of cerebral hæmorrhage almost always come from the branches of the middle cerebral artery. The middle lobe, the island of Reil, the corpus striatum, and the optic thalamus, are nourished through this vessel, and hence the great preponderance of extravasation in these portions of the encephalic mass.

---

[1] "Note sur la distribution des artères nourricières du cerveau." *Mouvement médical*, 1873, p. 27. Also, "Recherches anatomiques sur la circulation de l'encéphale." *Archives de physiologie*, 1874, p. 316.

[2] "Traité philosophique de médecine pratique." Paris, 1838, tome i., p. 448.

It has also been observed—and Durand-Fardel[1] calls special attention to the circumstance—that cerebral hæmorrhage has a manifest tendency to be developed and directed, rather toward the central than the peripheral parts of the brain. It is thus, to say, centripetal in its course, in which respect it differs from cerebral softening, which is not less evidently centrifugal—the peripheral regions showing a greater tendency than the central to be affected by this morbid process.

The right side of the brain appears to be more frequently the seat of cerebral hæmorrhage than the left. Thus, on consulting Gintrac,[2] we find that in three hundred and sixty-nine cases in which the side on which the lesion was situated was noted, the parts were affected in the order of frequency shown in the following table:

	Right.	Left.
Corpus striatum, optic thalamus, and these bodies simultaneously	73	63
Middle lobes	63	52
Pons Varolii	10	10
Cerebellum	14	12
Cortical substance	15	8
Posterior lobes	18	15
Anterior lobes	6	10
Total	199	170

The right side had thus a numerical superiority of twenty-nine over the left. It will be observed, also, that in no one part did the left side predominate except in the case of the anterior lobe. On the other hand, Durand-Fardel,[3] from an examination of one hundred and seventeen cases of hæmorrhage into the hemispheres, found that the right side was the seat in forty-nine, the left in fifty-seven, and both sides in eleven instances. Of eleven cases of cerebellar hæmorrhage, the right lobe was affected six, the left five times, and the middle lobe twice.

Generally there is but one recent extravasation, but occasionally two or more occur simultaneously, or at least so near to each other in point of time as to be essentially contemporaneous acts of one morbid process. Of one hundred and thirty-nine cases cited by Durand-Fardel,[4] twenty-one were multiple; eighteen of these were double, and three triple. In my own experience two cases of triple lesions have occurred, and two of double lesions. Of the triple cases the right corpus striatum, right middle lobe, and left middle lobe, were the seats in one, and the right and left corpora striata, and left anterior lobe, in the other. Of the double cases the seats in one were the right corpus striatum, and right middle lobe, and in the other the right middle and posterior lobe and right half of the pons Varolii.

It sometimes happens that the mass of extravasated blood breaks through the cortical substance of the brain, and appears immediately under the pia mater and arachnoid; or these membranes may give and the blood be effused into the space between them and the mater. In a very few of these cases the blood comes primarily the cortical substance of the brain, but in the greater number the travasation originates more deeply and reaches the surface by l ating the easily-torn white tissue. The blood in these cases under coagulation much more rapidly than when it remains in the cer substance, unless the base of the brain be the seat, in which case it remains fluid.

---

[1] "Traité pratique des maladies des vieillards." Paris, 1873, p. 181

[2] *Op. et loc. cit.* [3] *Op cit.*, p. 185. [4] *Op cit.*, p. 186.

The extravasation takes place into the ventricles in about one of all the cases. The lateral or fourth ventricle may be the sea it may exist in both of the former. The blood extravasated into ventricles remains liquid a longer time than when effused into other part. This is probably due to the fact that it is subjected t action of the ventricular fluid, by which its physical properties altered.

In the majority of cases of hæmorrhage into the ventricles, blood comes originally from the corpus striatum, or optic thala but it may also be derived from the choroid plexus, from the sep lucidum, or from the walls of the ventricles. Sometimes it is possible to determine its point of origin. It may enter the vent through a small opening, in which case the *foyer* is distinct, or wall of the ventricle may be largely lacerated and so broken down the *foyer* and the ventricle constitute essentially but one cavity. septum lucidum is not infrequently torn, and the two lateral vent are thus converted into one cavity.

As regards what may be called the secondary consequences o extravasation of blood into the cerebral substances, we find that it is large the convolutions are flattened against the walls of the cran the membranes are usually dry, and a distinct feeling of fluctuatio often be detected. In several cases I have known a large extravas to cause by its own weight a complete rupture of the lobe in whi existed, through the handling required in removing the brain from skull.

At other times the membranes are evidently congested; the b tissue, when incised, exhibits an increased number of red points, and subarachnoidean or ventricular liquid may be largely augmented the normal quantity.

The state of the arteries is a most important and interesting sub for examination, but, as it has an immediate and direct relation the pathogeny of cerebral hæmorrhage, it will be more properly co ered under the head of pathology.

Extravasated blood undergoes certain changes. Instead of viding into two parts, the clot and the serum, as does blood exposed to the atmosphere, it remains for a time homogeneous gelatiniform. About the fifth or sixth day it separates into two parts: the one, the serum, is absorbed by the surrounding tissue; the other consisting mainly of the fibrine and the red corpuscles, contracts and becomes hard. By the fifteenth day it has become fibrinous in texture, and is changed from its former black hue to a yellow color. Microscopic examination, made at any period during these changes, reveals the presence of red corpuscles, crystals of hematoidin and sometimes of cholestrin. It never entirely disappears.

In the earlier period of the extravasation, the walls of the cavity are rough, and discolored with blood. But, as the changes are going on in the clot, the walls likewise alter in appearance; the inequalities and irregularities disappear, and a new formation of connective tissue lines the cavity. Blood-vessels appear in it, and aid in the absorption of the fluid portion of the extravasated blood. As the process of separation and absorption goes on, the cavity contracts upon its contents, and eventually forms a cicatrix which incloses the remains of the clot. This cicatrix is generally of a yellow color, and firm in texture.

Sometimes, however, absorption does not take place. The contraction of the walls of the cavity does not therefore ensue, and it remains distended with more or less altered blood. This may be the starting-point of secondary lesions, or a new hæmorrhage may occur into the same cavity, or an abscess may result.

Pathology.—The theory of cerebral hæmorrhage brings us to the consideration of several important points. One of the first questions to be solved is, Can the rupture of a vessel of the brain take place—not including traumatic causes—unless the vessel is in a diseased condition? Both sides of this proposition have their adherents. On the one part, it is urged that cerebral hæmorrhage never takes place spontaneously unless the walls of the bleeding vessel have been so injured by disease as to destroy their strength and elasticity; on the other, that it is perfectly possible for a blood-vessel to give way, owing to the increased tension of the blood or disease of the perivascular tissue, without the walls of the vessel itself being in the least diseased. While admitting that, in the majority of cases, the structure of the yielding vessel will be found to be impaired, I am satisfied that either of the other two causes may produce a rupture. The reasons for this opinion will be apparent in the course of the following remarks.

One of the most common diseases to which the cerebral arteries are liable is, chronic endarteritis, a condition which has been well described by Virchow,[1] and which is particularly apt to be met with in those who, from age or other debilitating influence, have had their nutrition impaired. As the consequence of this state, the vessels lose their elasticity, become brittle, and are therefore often unable to bear the ordinary tension of the blood, much less any severe strain. This disease may terminate in fatty degeneration of the arterial walls, or this last condition may be the primary affection. Fatty degeneration, like chronic endarteritis, is most commonly met with in badly-nourished persons, but who are at the same time cachectic. The inner coat is the point of origin, and hence it sometimes happens that this and the middle coat give way, leaving the external coat entire, and thus forming an aneurism. But Bouchard,[1] who has examined into this matter with great minuteness, denies that such aneurisms are ever found, and asserts that the so-called aneurismal sac consists of the lymphatic membrane, lining the cavity in the perivascular tissue, through which the vessel passes; and that the blood, in such cases, has already ruptured the vessel. In reality, however, there is no hæmorrhage into the cerebral tissue till this membrane gives way.

---

[1] "Ueber die Erweiterung kleinerer Gefasse." "Archiv für Path. Anat. und Physiol.," B. III., 1848, and "Cellular-pathologie," Berlin, 1871, S. 458, *et seq.*

In a subsequent memoir, by MM. Charcot and Bouchard,[2] this point is still more thoroughly considered, and the opinion expressed that cerebral hæmorrhage is almost invariably due to what they call miliary aneurisms, which are the result of arteritis, and which are not necessarily preceded by atheroma.

The existence of these minute aneurisms was first pointed out by Cruveilhier,[3] and was subsequently recognized by Calmeil.[4] Meynert[5] appears also to have noticed them, and Heschel[6] discovered them in the pons Varolii; but no one previous to Charcot and Bouchard called attention to the relation which they bear to cerebral hæmorrhage. On March 16, 1866, while examining the *foyer* of a recent extravasation into the brain, they perceived, on the walls of the cavity in the cerebral tissue, two small globular masses attached to a minute vessel. These were miliary aneurisms. One was ruptured, and its contents were in immediate relation with the mass of extravasated blood constituting the apoplectic clot. Previously to this time these observers had noticed these aneurisms, but not before had they associated them with the pathogeny of cerebral hæmorrhage; since then, in numerous communications, they have called attention to the importance of their discovery, and its value is generally acknowledged by neuro-pathologists. In the accompanying woodcut (Fig. 10), taken from Bouchard's memoir, is represented one of these aneurisms which has been ruptured into a hæmorrhagic clot: *a*, the aneurism; *b*, the clot; *c*, *c*, the torn perivascular or lymphatic sheath.

---

[1] "Études sur quelques pointes de la pathogénie des hémorrhagies cérébrales." Paris, 1866.

[2] "Nouvelles récherches sur la pathogénie de l'hémorrhage cérébrale." *Archives de physiologie normale et pathologique*, 1868, pp. 110–643.

[3] "Anatomie pathologique du corps humain," liv. xxxiii., Pl. 2, Fig. 3.

[4] "Traité des maladies inflammatoires du cerveau." Paris, 1859, tome ii., p. 522.

[5] "Ueber Gefassentartungen in der Varolsbrücke und den Gehirnschenkeln." *Allgemeine Wiener Wochenschrift*, No. 28, 1864.

[6] "Die Capillar-Aneurysmen im Pons Varolii." *Wiener Medicinische Wochenschrift*, September, 1865.

FIG. 10.

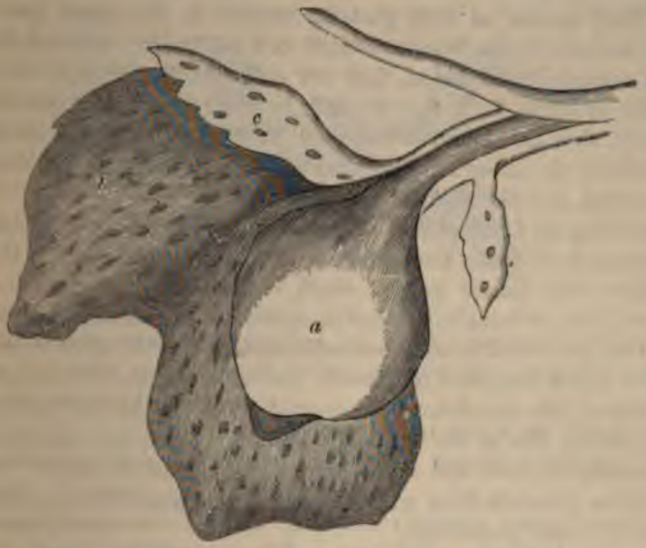
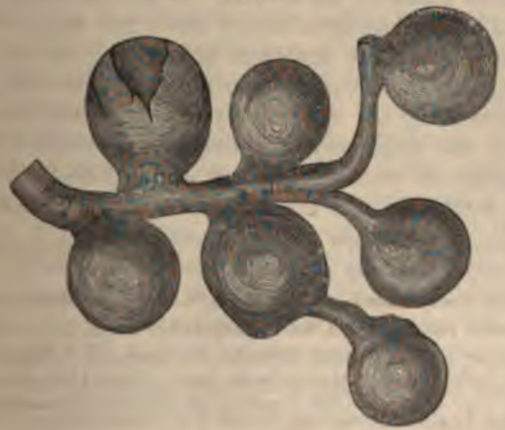


Fig. 11 is from the drawing of a vessel which I recently dissected out of the pons Varolii, into the right lobe of which a large extravasation had taken place. Both lobes were studded with these aneurisms; they were also found in the convolutions in the optic thalami and cor-

FIG. 11.



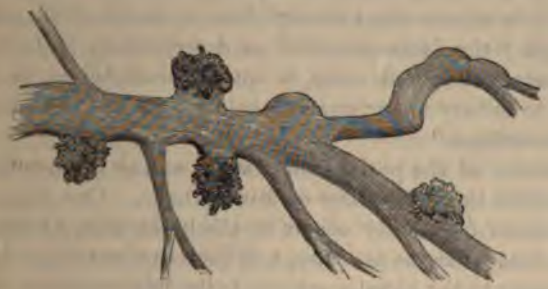
pora striata, and in the white substance of both hemispheres; a large extravasation had also taken place into the right hemisphere.

In sixty-nine cases of cerebral hæmorrhage in which post-mortem examinations were made, atheroma was found but in fifteen, or twenty-two per cent., while these miliary aneurisms were met with in every case. They appear as little globular masses in the small intracranial vessels, and are in size from one-tenth of a millimetre to one millimetre. If they contain liquid blood, they are red; but, if the blood be coagulated, the color is dark, almost black in some cases. In the order of frequency, they are found in the optic thalami, the corpora striata, the convolutions, the tuber annulare, the cerebellum, the centrum ovale, the crura cerebri, and the medulla oblongata.

According to Charcot and Bouchard, the arteritis, which results in the formation of these aneurisms, is diffuse in character. It is found not only in the minute artery, which is the subject of the aneurismal dilatation, but extends to the entire system of minute intracranial vessels. This arteritis is in some respects analogous with what Rokitansky described under the name of chronic peri-arteritis, and is characterized by disease of the membrane, designated by Robin as the perivascular sheath, and by His as the lymphatic sheath. There are also lesions of the adventitious tunic and of the muscular and internal coats. The diseased action proceeds from without inward, and hence the name of peri-arteritis is a very proper one.

Charcot and Bouchard claim that, with the following exceptions, all cases of cerebral hæmorrhage are the result of the rupture of miliary aneurisms, viz., fracture with depression; the hæmorrhages which result from thrombosis of the sinuses, and those which occur in the course of certain depraved states of the system. While admitting that the majority of cases of cerebral hæmorrhage have this origin, I am not prepared to go so far as these observers in ascribing all not embraced in the three categories of exceptions above specified, as being due to this cause. I had recently the opportunity of convincing myself that this explanation of the pathogeny of cerebral hæmorrhage is too absolute; for, on examining the brain of a patient who had died from an extravasation of blood into the left corpus striatum, optic thalamus, and left lateral ventricle, not a single miliary aneurism could be discovered, although they were carefully sought for in all parts of the brain. The patient, a lady forty-three years of age, had suffered from repeated attacks of acute rheumatism, had frequently been affected with headache and vertigo, and had been seized with apoplexy while in the water-closet. She had been the subject of heart-disease for over twenty years. I had only the brain submitted to me for examination, but all the arteries of this organ were in a state of atheromatous degeneration, and I was able to find what appeared to be the vessel, or one of them, which had given way and produced the extravasation. The accompanying engraving (Fig. 12) represents this artery as seen with an inch-objective. It is perceived that several of the aneurismal dilatations have given way; the internal coat of this, as well as of other arteries, was found, by microscopical examination, to be in a state of fatty degeneration; the same state existed in the middle coat, and the external coat was thickened and friable.

FIG. 12.



Lancereaux[1] reports a very similar case, of which, as it has an important bearing on the subject, I quote the summary which he gives (page 424):

"Hæmorrhage into the left" [right is evidently meant, and it is so stated on page 252, where the full report of the case is given] "corpus striatum, producing an irruption into the lateral ventricles, and arteritis, albuminuria, cardiac hypertrophy.

"A woman, aged fifty-eight, died a few days after an attack characterized by left hemiplegia, diminution of sensibility, and vomiting. The autopsy revealed the existence of a hæmorrhagic clot at the exterior and posterior part of the corpus striatum, which, after having separated this ganglion from the optic thalamus, had broken into the ventricular cavity. The nervous tissue, besides being torn, was colored yellow, through the infiltration of hæmatine into its substance. The ventricles contained a small quantity of liquid blood. There existed under the ependyma of the posterior cornu of the right ventricle a hæmorrhagic punctation, and a sanguineous suffusion extended over the whole circumference of the cerebellum. The entire encephalic mass was injected. The walls of the cerebral arteries were thick and opaque. On the branches, even those of the smallest size, were perceived moniliferous dilatations, the result of a primitive alteration of the arterial wall, and the probable points of origin of the hæmorrhage. The aorta was affected with endarteritis throughout its whole extent, the aortic orifice was slightly insufficient, and the left ventricle was markedly hypertrophied; the renal arteries were indurated, rigid, and calcareous. The kidneys, small, atrophied, and granular, were affected with interstitial nephritis. The arterial system was involved throughout almost its entire extent."

---

[1] "Anatomie pathologique," texte, pp. 252 and 424; atlas, plates 24 and 43.

It would appear, therefore, that we cannot set aside the results obtained by Virchow and others, and that, in the present state of our knowledge, it is safe to adopt the opinion expressed by Durand-Fardel,[1] that, although "the facts observed as described by MM. Charcot and Bouchard have undoubted value, it would, nevertheless, be premature to attribute to miliary aneurisms an exclusive part in the production of cerebral hæmorrhage."

The condition of the perivascular tissue, or the brain-substance, has much to do with the occurrence of hæmorrhage. One reason why extravasation more frequently occurs in the brain than in the liver, for instance, is, that its tissue is softer, and therefore not capable of giving as much support to the blood-vessels as is the latter organ. Now, when the cerebral substance is softened by disease in any part, the natural support of the vessels of that part is still further lessened, and the tendency to hæmorrhage increased. Again, in the condition sometimes met with in old people, in which the brain becomes atrophied, the vessels may undergo dilatation and subsequent rupture. This view is opposed by Jaccoud,[2] but in one case of cerebral hæmorrhage, terminating in death, and in which I had the opportunity of making a post-mortem examination, the right hemisphere, the seat of the extravasation, was very considerably atrophied, and weighed three ounces and a quarter less than the left. The possibility of the existence of this cause may, therefore, be admitted, although it cannot be considered as definitely established. The researches of Cotard[3] would appear to show that cerebral hæmorrhage is not infrequently a cause of partial atrophy of the brain.

In the next place, the state of the blood, as regards quality and tension, must be considered. There can be no doubt that certain diseases affecting the general system may so deteriorate the blood as to render it unfit to properly nourish the blood-vessels, and hence their tissue is more readily broken down. Among these conditions are typhus, scurvy, chlorosis, gout, and syphilis.

The tension of the blood in the vessels is subject to constant variation from the operation of many physical and mental causes, and may, through their action, be so increased as to overcome the resistance afforded by the vascular walls. These influences have been sufficiently considered in the section on causes, and need not, therefore, be dwelt upon here at any length. My own opinion of their sufficiency, without preëxisting disease of the blood-vessels, to produce rupture and extravasation, has been formed after much observation and reflection.

---

[1] *Op. cit.*, p. 262.

[2] *Op. cit.*, p. 155.

[3] "Étude sur l'atrophie partielle du cerveau," Paris, 1868.

Analogous phenomena take place every day, and are not supposed to be due, in any extent, to vascular disease. Thus nasal hæmorrhage occurs from strong muscular exertion of such a character as to retard the flow of blood from the brain, from emotional or other kind of mental excitement, and from hypertrophy of the left side of the heart, by which the amount of blood in the cerebral vessels is increased. All these causes augment the tension, and it would be singular if at times a healthy intracranial vessel did not give way through their influence, as well as one outside of the skull.

Differential Diagnosis.—A point of very great importance remains to be considered as a part of the pathology, and that is whether it is possible or not to determine during life in what part of the brain an extravasation has taken place? While I am afraid we cannot be as explicit in this matter as is desirable, I am very sure we can often, from a careful study of the symptoms, arrive at conclusions more or less accurate, and can sometimes determine the question with absolute certainty. The great difficulty is, that we are not yet sufficiently acquainted with the physiology of the several parts of the brain, and hence are not able to ascribe, with as much sureness as is desirable, variations from healthy action, to derangement of the proper anatomical part of the cerebral mass. Besides, when the extravasation is large, although it may be strictly confined to the anatomical limits of the ganglia or part of the encephalic mass in which it originates, it may act by transmitted pressure upon contiguous ganglia or parts, and hence the symptoms are rendered complex.

As we have seen, hæmorrhage is more liable to take place within the ganglia constituting the motor tract than any other part of the brain. This is mainly due to the fact that this is the most vascular part of the cerebral substance.

When the lesion is limited to either the intra-ventricular or extra-ventricular nucleus of the corpus striatum of one side, the speech is generally affected from paralysis of the tongue, and there is loss of the power of voluntary motion on the opposite side, but no abolition of sensibility, except, perhaps, for a few hours. Cases in illustration of this fact have been given by Andral[1] and Luys,[2] and one instance in my own experience was established by post-mortem examination. The patient, a man of sixty-two years of age, had been hemiplegic for eleven years, and died suddenly, in April, 1851. Post-mortem examination showed the cause of death to have been fatty degeneration of the heart. On examining the brain, a cicatrix was discovered in the right corpus striatum. The hemiplegia was on the left side, and had never been accompanied with any loss of sensibility. There was no other lesion of the brain, so far as could be ascertained.

---

[1] "Clinique médicale," tome v., pp. 319–321, 442.

[2] "Recherches sur le système nerveux cérébro-spinal," etc., p. 545.

Generally speaking, when the clot is strictly limited to either of the nuclei of the corpus striatum, the paralysis, however extensive it may have been in the first place, is of a transitory character. Moreover, there is no tendency to the production of muscular contractions at a late period of the disease.

And there are instances on record in which there has been extravasation into the corpus striatum, and no paralysis of any part of the body. Gintrac,[1] of forty cases collected by him, found apparent absence of paralysis in five. But he admits that this number may perhaps be reduced, for one of the cases was that of an infant one day old, and the other, that of an old man eighty years of age, who had had a cerebral hæmorrhage ten years before his death, in both of which an exact diagnosis of this point could not have been otherwise than difficult. But in one of the others there was no paralysis, and yet after death a clot as large as a pigeon's egg was discovered in the left corpus striatum. In the second there was no actual paralysis, but a weakness and trembling of the right arm. The post-mortem examination revealed the existence of a clot, as large as an almond, in the left corpus striatum. The third was for a few moments deprived of the power of speech, but he had equal muscular strength on both sides. Then he became weak and died, without having been actually paralyzed. After death a cavity filled with a brown serous fluid was found in the anterior and external part of the right corpus striatum, and the whole of the left posterior lobe was reduced to a yellowish pulp, and was studded with purulent *foyers*. This was certainly not an uncomplicated case. And thus of the five there was but one in which there was indubitably no paralysis.

The optic thalamus is another common seat of extravasation. In such a case the observed symptoms are especially connected with the organs of the special senses. Thus there are double vision, dilatation or convulsive movements of the pupil, blindness, and anæsthesia or hyperæsthesia of the paralyzed parts of the body. As in lesion of the corpus striatum, the paralysis of motion, if present at all, is on the opposite side of the body, and is usually transient in character. The hearing and smell may also be affected. Luys[2] has collected a large number of cases in support of the view here enunciated.

The researches of Virenque[3] also go to show that lesions of the optic thalamus are accompanied with loss of sensibility on the opposite side of the body. His observations, therefore, are entirely confirmatory of those of Turck,[4] who in four very carefully recorded cases found hemi-anæsthesia coexistent with lesion of the optic thalamus and corpus striatum of the opposite side.

---

[1] *Op. cit.*, tome ii., p. 142, *et seq.*

[2] *Op. cit.*, p. 534, *et seq.*

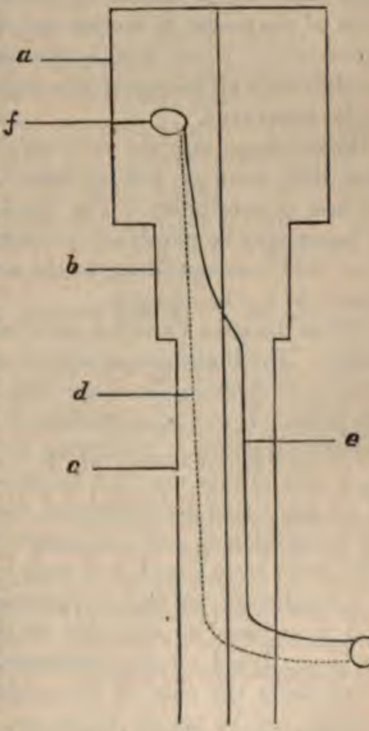
[3] "De la perte de la sensibilité générale et spéciale d'un coté du corps (hémianæsthesiæ) et de ses relations avec certaines lésions des centres opto-striés." Paris, 1874.

[4] "Ueber die Beziehung gewissen Krankheitsherde des grossen Gehirns zur Anæsthesie. Sitzungsberichte des Kais. Kön. Academie der Wissenschaften, B. xxxvi., 1859.

In those cases of cerebral hæmorrhage limited to the optic thalamus, paralysis of motion when it exists is less intense than when the corpus striatum is also involved, and is often restricted to the inferior limbs. The speech is rarely involved.

The intelligence is not notably lessened, but there is often a marked proclivity to the supervention of hallucinations of the special senses. This is not surprising when we take into consideration the fact that recent physiological observations appear to show that the optic thalami are centres for the special senses. Luys[1] has very thoroughly worked up this subject,[2] and Ritti has recently in a philosophical essay adduced many facts and arguments to show the relations of lesions of the optic thalamus with hallucinations. In thirty-two cases of hallucinations, mainly of the sight and hearing, but sometimes of all the senses, post-mortem examinations revealed the existence of some kind of lesion of the optic thalami.

FIG. 13.



It generally happens that an extravasation, originating in either the corpus striatum or optic thalamus, involves both these ganglia. Hence we have, as the most common symptoms of hæmorrhage into these organs, loss or impairment of the power of motion, disturbance of sensibility, dilatation or irregular movements of the pupil, aberrations of vision and hearing, etc.

As we have seen, a lesion of the corpus striatum and optic thalamus of one side produces loss of the power of motion and of sensibility in the opposite side of the body. The manner in which this is accomplished will be readily understood from an inspection of the accompanying diagram (Fig. 13), in which *a* indicates the left opto-striated body, *b* the left half of the pons Varolii and medulla oblongata, *c* the left lateral half of the spinal cord, *d* a sensory nerve-fibre decussating soon after its entrance into the cord,

---

[1] *Op. et loc. cit.*

[2] "Théorie physiologique de l'hallucination." Paris, 1874.

8

e a motor nerve-fibre decussating at the lower boundary of the medulla oblongata. A lesion existing at *f* will therefore cause paralysis of motion and of sensibility at *g*, on the right side of the body.

When the extravasation beginning in the left optic thalamus or corpus striatum extends to the fissure of Sylvius so as to involve the posterior part of the third frontal convolution, the island of Reil, or other part supplied by the middle cerebral artery, or when it originates in this region, aberrations of speech occur. These are independent of paralysis of the tongue, and are such as are embraced under the term aphasia. This subject will be hereafter more fully considered.

If the lesion be limited to the anterior two-thirds of the internal capsule, there will be merely paralysis of motion, although there may be, as I have lately had occasion to know, slight and temporary hemianæsthesia. If, however, the posterior third of the internal capsule be the seat of the hæmorrhage, there will be well-marked hemianæsthesia. Of course there are in almost all cases various proportional combinations of loss of the power of motion and of sensibility according to the exact position of the lesion in the internal capsule. And it generally happens that with all lesions of this region late contractions of the opposed muscles supervene.

Hæmorrhage into the crus cerebri produces hemiplegia of the opposite side, more or less extensive, according to the size of the clot, with loss of sensibility. The third pair arises in part from the crus, and hence may be paralyzed, producing ptosis and external strabismus on the side corresponding to the seat of the lesion, and consequently opposite to the hemiplegia.

When the pons Varolii is affected, the crossed paralysis is still more marked. The limbs are paralyzed on the opposite side, and the face in whole or in part on the same side as that in which the hæmorrhage takes place. If the extravasation is in the mesial line, both sides of the body are paralyzed. According to Trousseau,[1] however, crossed paralysis is not always due to a lesion of the pons, as asserted by Gubler,[2] and as supported by additional cases collected by Luys.[3] Trousseau rests his opinion on one case, in which after death very extensive lesions of the brain were found, but none involving the pons.

Nevertheless we find in practice that when an extravasation of blood is confined to one side of the pons, and is not extensive, the face is paralyzed on the corresponding side. The facial nerve makes its exit from the side of the medulla oblongata; some of its roots of origin can be traced as far as the floor of the fourth ventricle, others come from the lower part of the medulla oblongata, and others descend from the upper border of the pons, where they probably decussate. Now, a lesion existing in a lateral half of the pons will, therefore, produce a paralysis of the corresponding facial nerve, and of the opposite spinal nerves; whereas, if it occur above the point of decussation of the encephalic fibres, the paralysis will be on the opposite side for all parts of the body. These facts are shown in the accompanying diagram (Fig. 14).

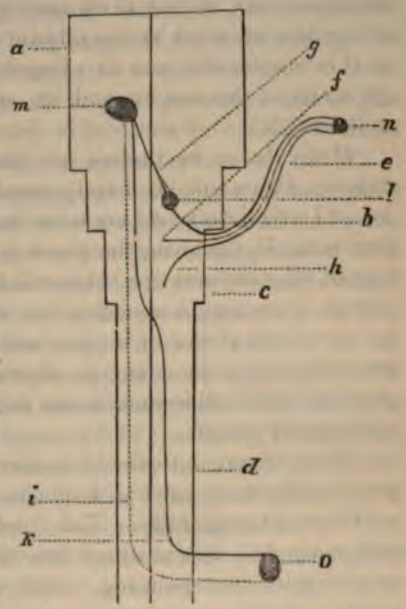
---

[1] "Lectures on Clinical Medicine," Bazire's translation, Part II., p. 333.

[2] "Sur l'hémiplégie alterne," *Gaz. hebd.*, October, 1856, and "Mémoire sur les paralysies alternes," etc., *Gaz. hebd.*, 1859.

[3] *Op. cit.*, p. 529, *et seq.*

Fig. 14.



a, the left hemisphere; *b*, right half of pons; *c*, right half of medulla oblongata; *d*, right half of spinal cord; *e*, right facial nerve; *f*, fibre of origin from nucleus in medulla oblongata; *g*, descending fibre decussating at upper border of pons; *h*, ascending fibre; *i*, sensory root of spinal nerve; *k*, motor root of sensory nerve; *l*, lesion in pons; *m*, lesion in left hemisphere; *n*, paralyzed part supplied by facial; *o*, paralyzed part supplied by spinal nerve.

It is obvious, from a study of this diagram, that a lesion of one lateral half of the pons (at *l*) will cause paralysis of motion and of sensibility of the opposite side of the body generally, and of the corresponding side of the face; and that a lesion of the hemisphere (at *m*) will produce paralysis of the opposite side of the face and the body.

It is true that it is not definitely settled by histological investigation that the decussation of the ascending roots takes place, but pathology is just as capable of determining the question as histology. Vulpian[1] asserts that the decussation of the roots of the facial occurs in the mesial line of the medulla oblongata at the junction of the two nuclei of origin; but, if this were the case, a lesion of one side of the pons would necessarily be followed by double facial paralysis, a sequence which does not in reality ensue.

From the contiguity of the pons to the medulla oblongata, an extravasation of blood into it is generally accompanied by the symptoms which result from hæmorrhage into this latter organ, though they are not as a rule so strongly marked.

The principal phenomena indicating the medulla oblongata as the seat of extravasation are, loss of the power of swallowing, from paralysis of the glosso-pharyngeal, difficulty of protruding the tongue, from paralysis of the hypoglossal, and huskiness of the voice, tumultuous action of the heart, dyspnœa and gastric derangements, from paralysis or the pneumogastric nerve. There is in addition paralysis of one or both sides of the body.

---

[1] "Essai sur l'origine de plusieurs pairs de nerfs craniens. Thèse de Paris," 1853, p. 32.

An extravasation into the cortical substance of the cerebrum is characterized by no very definite aggregation of symptoms. There may be delirium, coma, disorders of speech, convulsions, paralysis, contractions or rigidity of either the paralyzed or sound limbs, vomiting, derangement of respiration, and occasionally anæsthesia or hyperæsthesia. Paralysis when present is upon the opposite side of the body from that of the lesion.

When the extravasation is in the white substance of the cerebrum, there may be no marked symptoms of diagnostic value. I have known cases in which large *foyers* have been formed with no other symptoms than intense pain in the head and persistent vomiting. But when blood is extravasated into the white tissue the quantity is ordinarily great, and as a consequence there are often symptoms present which are due to resultant pressure upon other portions of the encephalic mass. Thus there may be coma, paralysis, loss of sensibility, stertorous respiration, and other phenomena indicating derangement of the motor and sensory ganglia. The passage of the extravasated blood into the ventricles almost invariably causes contractions or convulsions of the muscles of the opposite side of the body.

The researches I have made[1] relative to the functions of the cerebellum would seem to show that its office is not materially different from that of the cerebrum. Still, I think there are some indications which, although not perhaps giving us the right to form a definite conclusion, are yet sufficiently well marked to enable us to arrive at a probable diagnosis between hæmorrhagic lesion of the cerebrum and that of the cerebellum. Thus, vertigo is almost an invariable accompaniment of the cerebellar extravasation ; vomiting is much more generally met with than when the cerebrum is affected ; hemiplegia is not so common ; the sensibility is never disturbed ; and the pain is in the back of the head.

Ferrier[2] has very clearly shown that irritation of the cerebellum produces nystagmus and defective power of ocular coördination. But I am not aware that these phenomena have been noticed in cases of cerebellar hæmorrhage. Hillairet,[3] in his excellent memoir, does not mention them as features of the affection. He distinguishes two forms of this lesion. In the one, the onset is sudden, and death soon follows; in the other, the course of the affection is slow, and life may be prolonged for a considerable period. In this latter, vomiting is a prominent feature. Hemiplegia, according to him, is always crossed. Sensibility remains unaffected till near the close of the disease by death, and there are no convulsions. The speech is not often affected. The special senses he did not find notably deranged, except in the last stage. In this result he differs with several other writers on the subject.

---

[1] "The Physiology and Pathology of the Cerebellum." *Quarterly Journal of Psychological Medicine*, April, 1869.

[2] "Experimental Researches in Cerebral Physiology and Pathology." "West Riding Lunatic Asylum Reports," vol. iii., 1873, p. 69, *et seq.*

[3] "Hémorrhagie cérébelleuse," *Annuaire de médecine et chirurgie pratiques*, 1859, p. 39. Also *Archives de médecine*, 58.

Besides a number of cases, some of which are referred to in the memoirs cited, one has occurred in my experience, in which I had the opportunity of making a post-mortem examination.[1]

A man had suffered from vertigo, occasional convulsions, attacks of nausea, and vomiting, and a constant and violent pain affecting the back of the head. The symptoms had ensued in consequence of a severe blow which he had received on the back of the head, by raising himself too soon while the horse he was riding was passing under a low archway.

When this man attempted to walk, he reeled and staggered as if he were drunk. The upper extremities and the organs of speech were not affected; he had the entire control of his legs when lying down, and there was no diminution of sensibility anywhere. At last, he became paraplegic, and shortly afterward died in a convulsion. The post-mortem examination showed the existence of an abscess which had obliterated nearly the whole of the left lobe of the cerebellum. The other parts of the brain were, so far as could be perceived, perfectly healthy.

Besides the occurrence of local secondary lesions, the immediate results of the presence of a foreign body in the cerebral tissue, there are others, which are due to the interruption of the normal brain-functions, which hæmorrhage so generally induces. Thus, atrophy of the cerebral structure may result, as has been pointed out by Cotard[2] and others, or the degeneration may extend to the spinal cord, as is so well shown by Bouchard.[3] In this latter event the process does not begin till about the end of the fourth or fifth month. It is mainly characterized by the supervention of permanent contraction of certain of the paralyzed muscles, and will be more appropriately considered under another head.

Another point in connection with cerebral hæmorrhage requires further elaboration. It is well known that the facial paralysis resulting from ordinary cerebral hæmorrhage is less extensive and less thoroughly marked than when it is due to disease or injury of the trunk of

[1] *Op. cit.*, p. 209.

[2] "Étude sur l'atrophie partielle du cerveau," Paris, 1868.

[3] "Des dégénérations secondaires de la moelle épinière,' *Archives gen. de médecine*, 1866. Also Hun's translation, *American Journal of Insanity*, 1869.

the seventh pair or to lesion of the pons Varolii. Thus we have seen that, in the former affection, the orbicularis palpebrarum escapes paralysis,[1] and the other muscles supplied by the facial nerve are usually not so profoundly paralyzed as when the pons or the nerve is the seat of the disease.

Many explanations have been offered of this remarkable circumstance, but the one given by Landry[2] is more nearly reconcilable with the anatomy and physiology of the parts involved than any other.

The nucleus of the facial is entirely comparable to the anterior cornua of the cord. It constitutes a little special motor nerve-centre which possesses a certain amount of autonomy. It is through this centre that the muscles of the face are directly made to contract. The encephalic fibres which connect it with the brain are only at the service of the psychical department, and an impulse sent through them is not of itself capable of exciting contraction in the muscles to which the facial is distributed. But, with the spinal cord, this nucleus possesses reflex excitability, and, as is the case in diseases of the brain in which the anterior columns suppress voluntary movements without destroying the reflex manifestations of which the gray substance of the cord is susceptible, so the cerebral lesion leaves to the nucleus of the facial the power to determine reflex contractions. It therefore continues to be excited by sensitive excitations which reach it from the periphery. Thus, in facial hemiplegia of cerebral origin, we observe, from time to time, certain movements which appear to be voluntary because the provocative sensitive impression, which may only consist of the contact of air, remains unperceived. Accordingly, the orbicularis palpebrarum appears, above all the other muscles, to preserve its mobility, for its movements are principally excited by the stimulus of the light, which the lesion of the cerebral lobes does not prevent being reflected to the nucleus of the facial. In extensive diseases of the pons, however, the nucleus of the facial, situated as it is, in immediate proximity to this organ, is almost always compromised with it. In such a case, therefore, both reflex excitability and voluntary power are destroyed, and the paralysis is complete.

Treatment.—The means of treatment in cerebral hæmorrhage are, first, those which are applicable to the prodromatic stage, with a view of preventing any lesion; second, those proper during the seizure; and, third, those which are to be directed against the consequences of an attack.

---

[1] Bazire, in his translation of Trousseau's "Clinical Lectures," calls attention to the fact that, in ordinary cases of cerebral hæmorrhage, the patient, though able to close the eye of the affected side, cannot do so without, at the same time, closing the other, a fact which shows some loss of power. Since my notice was directed to this circumstance, I have observed that the patient is often sensible of the fact that the eye of the affected side cannot be closed as strongly or as rapidly as the other eye.

[2] Quoted by Poincaré, "Leçons sur la physiologie normale et pathologique du système nerveux," tome deuxième, Paris, 1874, p. 55.

It often happens that an attack may be prevented, even where the threatenings are very decided. The condition of the brain is such that the indications are to lessen the tension of the blood as much as possible. As I have already remarked, under the head of cerebral congestion, the bromides of potassium and sodium are peculiarly efficacious in accomplishing this end. Lately, in consequence of the investigations of Dr. S. Weir Mitchell, of Philadelphia, I have made much use of the bromide of lithium in cerebral congestion with or without a tendency to hæmorrhage, and have reason to prefer it to either the potassium or sodium salt. One feature of its action, which renders it especially useful in such cases as those now under notice, is the short interval which elapses between its administration and the effect. I am very sure I have given it successfully in several cases in which the bromides mentioned would not have acted so happily. In one of these, a gentleman from the South, who had already had an attack, and who was in consequence hemiplegic, was relieved of his vertigo, headache, numbness, and thickness of speech, by one dose of thirty grains, in less than half an hour. The bromide of calcium, a compound to which I have recently called attention,[1] is still more eligible. It acts more rapidly than any of the other bromides, and may be given for a longer period with less derangement of the organism. The dose is from fifteen to thirty grains, or even more, if only a single dose is to be administered. The oxide of zinc may also be given with advantage.

The bowels, if costive, should be opened by a brisk purgative; the stomach, if overloaded, should be emptied by an emetic, during the action of which warm water should be freely drunk so as to obviate, as far as possible, all straining; muscular exertion should be avoided, the head should be kept cool and well elevated, and the mind in a state of the utmost tranquillity.

During an attack, and throughout the whole period of reparation of damages, the less that is done in the vast majority of cases the better. The question of the propriety of bloodletting will generally even yet arise, but should in nearly every case be decided in the negative. I say nearly, for I know of but one possible form of attack in which it can by any possibility not only not be useful, but fail to do harm; and that is in a strong, plethoric person, with a full, bounding pulse, in whom, from the gradual development of the symptoms, we have reason to suspect that the hæmorrhage is still going on. In such a case, six or eight ounces of blood may be taken from the arm. But, in the case of cerebral hæmorrhage, attended by coma and the ordinary symptoms of the apoplectic condition, there is nothing to be done in the way of medication which can afford the slighest prospect of relief. It is true, a patient thus situated may recover if his attack is not of the severest kind, but it is not through any medicines we give him. Correct views relative to this point are far from being prevalent, and can only be established by regard being paid to the morbid anatomy and pathology of the subject.

---

[1] Note relative to Bromide of Calcium. *New York Medical Journal*, December, 1871, p. 591.

A clot in the brain is, to all intents and purposes, a foreign body, and both it and the walls of the cavity must undergo certain fixed and definite changes. In order that these changes may go on with the utmost possible regularity and certainty, all the powers of the system are requisite. The processes are not morbid ; on the contrary, they are in the highest degree conservative. To take blood from a body which is striving by all its agencies to repair an injury, is to deprive it of a portion of its strength without in the slightest degree accelerating the actions at the seat of the lesion. As Trousseau[1] remarks, no physician ever thinks of bleeding for an extravasation of blood under the skin, for he knows how perfectly absurd such a practice would be ; and yet, except as regards location, there is no difference between it and the cerebral clot. A prize-fighter, for instance, receives a blow in the face, which ruptures a blood-vessel and gives him a "black eye." He has an extravasation of blood into the cellular tissue. What would be thought of the physician who would recommend bloodletting from the arm, with a view of causing the absorption of the clot? The prize-fighter has found out by experience that he can open the skin with a knife, and let the blood out. The practice is excellent, and would be admirable for the brain also, were this organ of no more vital importance than the skin of the face. I have never bled a patient for cerebral hæmorrhage since 1849, and I am very sure that I have had no reason to regret the abandonment of the practice.

It is a common practice for purgatives to be given, and even so conservative a practitioner as Dr. J. Hughlings Jackson[2] puts "two drops of croton-oil on the tongue," why, he does not state, and certainly the practice is in direct antagonism not only with his assertion that "the chief thing is to keep the patient quiet," but with the general tenor of his theory of treatment. I have seen great annoyance and an aggravation of the symptoms from the indiscriminate administration of croton-oil. It is only, in my opinion, admissible when there is obstinate constipation, and when after three or four days the bowels have not been moved.

And then as regards iodide of potassium. There seems to be an idea prevalent that this substance exerts a powerful influence in cau ing the more rapid absorption of the extravasated blood, and hence it is frequently administered in large and frequently-repeated doses. I have often seen patients, at as early a period as possible, while still in a state of profound coma, dosed with the iodide of potassium to the extent of five grains every hour, with the object of causing the immediate absorption of the extravasated blood. That such a result is impossible no one acquainted with the morbid anatomy and the pathology of the subject will deny.

---

[1] "Lectures on Clinical Medicine," Bazire's translation, Part I., p. 10.

[2] Reynolds's "System of Medicine," vol. ii., article "Apoplexy and Cerebral Hæmorrhage," p. 541.

In fact, there is nothing to be done beyond keeping the patient perfectly quiet, with the head well elevated, and in a room, when possible, with a temperature of about 60° and thoroughly ventilated. Indications should be met as they arise. The bowels, if not moved naturally every day, may be emptied by an enema of warm water; the urine, if not passed by the patient, should be drawn off by the catheter; the strength, if feeble, as indicated by the pulse, should be kept up by the cautious use of stimulants; and, if the patient is restless and does not sleep well, some one of the bromides should be administered.

Ergot may, on theoretical grounds, be recommended in those cases in which we have reason to believe that the hæmorrhage is still going on; but I have no personal experience of its power in such instances. If administered, it should be given with no sparing hand.

The food should be of the most nutritious character, so as to be small in quantity, and should be taken frequently, day and night. Beef-tea, or the extract of beef, made according to Liebig's formula, supplies every indication.

If symptoms of inflammation make their appearance, cold applications may be made to the scalp, or a blister may be applied to the nape of the neck. Blisters or mustard-plasters to the wrist or ankles are absurd.

Nothing should be done for the relief of the paralysis till all signs of irritation of the brain have disappeared, and the patient begins to feel the restraint of confinement, and to make efforts to move his paralyzed limbs. These evidences of improvement generally begin soon after the eighth day. In about two weeks, therefore, it will be proper, in the majority of cases, to take active measures to restore the power of motion, and to prevent those contractions which tend to make a restoration much more difficult. The agents to be employed are passive motion, strychnia, phosphorus, and electricity. The first is accomplished by flexing and extending the joints of the affected limbs, by friction, and by kneading the muscles with the fingers. These movements should be performed every day for five or ten minutes at a time. The patient should likewise be encouraged to move the limbs by his own volition as often as possible short of causing fatigue. Strychnia should be given in doses of the one-twenty-fourth of a grain three times a day, or, preferably, by subcutaneous injection, in somewhat smaller doses once a day. In old cases of hemiplegia, the effects of strychnia thus administered are often well marked, and are exhibited when administration by the stomach has failed to produce a beneficial result. This is seen in the following brief abstract of sixteen cases which will serve as types of numerous others which have occurred in my private practice :

CASE I.—H. A., aged fifty ; male ; right hemiplegia. Came under treatment January, 1865 ; strychnia ineffectual by the stomach ; thirteen injections, of from one-thirty-second to one-twenty-fourth grain ; much improved.

CASE II.—J. S. ; forty-two ; male ; left hemiplegia. February, 1865 ; thirteen injections ; much improved.

CASE III.—S. T. ; sixty ; female ; right hemiplegia. February, 1865 ; strychnia ineffectual by the stomach ; nine injections ; much improved.

CASE IV.—I. S. ; sixty ; female ; right hemiplegia. April, 1865 ; five injections ; much improved.

CASE V.—M. T. ; fifty-two ; male ; right hemiplegia. April, 1865 ; strychnia ineffectual by the stomach ; eleven injections ; cured.

CASE VI.—O. S. ; sixty-three ; female ; left hemiplegia. April 30, 1865 ; secondary contractions ; twenty-two injections ; no improvement.

CASE VII.—B. R. ; forty-seven ; male ; left hemiplegia. June 11, 1865 ; strychnia ineffectual by the stomach ; seven injections ; much improved.

CASE VIII.—R. F. ; fifty ; male ; left hemiplegia. June 17, 1865 ; strychnia ineffectual by the stomach ; eight injections ; cured.

CASE IX.—T. W. ; forty-eight ; male ; left hemiplegia. September 5, 1865 ; eight injections ; much improved.

CASE X.—T. S. ; forty-nine ; male ; left hemiplegia. September 7, 1865 ; secondary contractions ; five injections ; no improvement.

CASE XI.—J. J. ; fifty-seven ; male ; left hemiplegia. September 11, 1865 ; secondary contractions ; no improvement.

CASE XII.—J. W. ; fifty-two ; male ; right hemiplegia, affecting arm only, at the time treatment was begun. September 27, 1865 ; strychnia ineffectual internally ; six injections ; cured.

CASE XIII.—W. M. ; forty-five ; male ; left hemiplegia. October 19, 1865 ; strychnia ineffectual internally ; seven injections ; cured.

CASE XIV.—S. M. ; forty-one ; male ; right hemiplegia. June 17, 1867 ; arm alone affected ; strychnia ineffectual by the stomach ; twenty injections ; cured.

CASE XV.—M. C. ; forty-four ; male ; right hemiplegia, affecting tongue and face only. July 1, 1867 ; ten injections ; so much improved as to be able to talk with fluency.

CASE XVI.—C. C.; fifty; male; right hemiplegia. May 4, 1869; strychnia ineffectual by the stomach; thirty-five injections; much improved.

Dr. Charles Hunter[1] has called attention to the advantages to be derived from the hypodermic use of strychnia in hemiplegia; and my former clinical assistant, Dr. R. A. Vance,[2] has adduced several cases to the same effect. Instances in support of the views above set forth occur daily in my private practice, and at the New York State Hospital for Diseases of the Nervous System. I have every reason, therefore, to be convinced of the good results to be derived from the practice.

Phosphorus administered in the form of phosphide of zinc, separately or in combination with the extract of nux-vomica, according to the formula given on page 54, is also a useful remedy.

But no agent is so valuable in hemiplegia as electricity, and amendment almost invariably follows its use, even in old cases, in which there are tonic contractions. If the case is seen soon after the seizure, the induced current will generally be sufficient to produce contractions of the paralyzed muscles. The poles, terminated by wet sponges, should be applied to the skin covering the muscles, or in some cases to the nerves. The current should be strong enough to cause slight pain, or, if sensibility is lessened, to produce contraction. In old cases attended with atrophy of the muscles, and diminished or abolished electro-contractility, the primary current may be necessary. It should be applied in such a manner as to be interrupted, for contractions are only caused when the circuit is closed and opened. As the muscles improve in size and irritability, the induced current should be used. Care should be taken not to fatigue the patient, or to cause excessive pain by employing a current of too great a degree of intensity.

As regards the restoration of sensibility, it will generally be found to be less difficult than the removal of the motor paralysis. The anæsthesia very often disappears or becomes much less spontaneously, and it does so from the centre to the periphery; that is, if there be anæsthesia of the leg, the sensibility returns in the upper part first, and subsequently in the lower part. The treatment consists mainly in the use of the electric wire-brush, which should be passed gently over the skin previously made dry. The other pole consists of a wet sponge. Either the induced or primary current may be used. If the latter, however, be employed, the wire-brush should constitute the positive pole.

I have frequently succeeded in curing almost complete anæsthesia from cerebral hæmorrhage by this treatment alone. In recent cases it will almost invariably prove effectual. Hyperæsthesia, if present, may be similarly managed. Mainly through the researches of M. Charcot and his pupils I have recently made use of strong magnets in the treatment of the hemiplegia apparently resulting from cerebral hæmorrhage. In one of these the patient recovered the power of motion, and the hemianæsthesia disappeared within less than half an hour; and in the other sensation was entirely restored to the hemianæsthetic side in less than five minutes. In both, a magnet capable of sustaining ten pounds of iron was laid against the paralyzed side of the body.[1] From experiments recently performed in France and in this country, it is extremely probable that there are other æsthesiogenetic factors—among these are various metals and woods and blisters. It is difficult, however, in the present state of our knowledge, to determine how much of the curative effect is due to the substance employed and how much to the "principle of suggestion."

---

[1] *British and Foreign Medico-Chirurgical Review*, April, 1868.

[2] *Journal of Psychological Medicine*, April, 1870. The first thirteen cases cited in this work were published in Dr. Vance's paper.

CHAPTER IV.

CEREBRAL MENINGEAL HÆMORRHAGE.

By the term cerebral meningeal hæmorrhage is to be understood—1. An extravasation of blood between the cranium and the dura mater; or, 2. An extravasation into the cavity of the arachnoid between the two layers of which this membrane is composed; or, 3. An extravasation into the sub-arachnoideal space between the arachnoid and the pia mater, or into the tissues of this latter membrane, or between it and the brain. There are thus—1. Extra-meningeal hæmorrhages; 2. Intra-arachnoideal hæmorrhages; and, 3. Sub-arachnoideal hæmorrhages. The first are almost always the result of traumatic cause, involving injuries of the cranium, by which the vessels of the dura mater are wounded. Extra-meningeal hæmorrhage may likewise be produced by the operation of trephining, should any of the vessels of the dura mater be divided.

It is, however, beyond doubt that this species may originate independently of wounds and injuries.

The distinction between intra- and sub-arachnoideal hæmorrhages was first pointed out by Prus,[2] to whom we are also indebted for much valuable information on the subject.

Of one hundred and seventy-two cases collected by Gintrac,[3] five were extra-meningeal, one hundred and nine intra-arachnoideal, and thirty-four sub-arachnoideal.

---

[1] "The Therapeutical Use of the Magnet." *New York Medical Journal*, November, 1880.

[2] "Mémoire sur les deux maladies connues sous le nom d'apoplexie méningée.—Mémoires de l'académie de médecine, tome xi., 1845, p. 18.

[3] *Op. cit.*, tome i., p. 732.

Symptoms.—The most prominent symptom of meningeal hæmorrhage is coma, which may appear suddenly, or be preceded by premonitory symptoms, such as headache, vertigo, and general convulsions. The stupor is usually profound, and does not differ from that observed in the severe forms of cerebral hæmorrhage. The power of motion is generally lost throughout the body, and consequently there is usually no hemiplegia. The reason for this is, that the hæmorrhage is so extensive as to press upon both hemispheres. Reflex and automatic movements remain, except when the medulla oblongata is involved, when some of them are abolished. If the extravasation is in this latter situation, death soon takes place from cessation of respiratory actions. Anæsthesia is present in the skin of those parts in which the power of motion is lost.

In ordinary cases the patient may pass out of the comatose condition from the fact of the brain becoming accustomed to the pressure, and he then may be able to speak, and to move his limbs, but his mental and physical faculties are greatly enfeebled, and a renewal of the hæmorrhage again plunges him into a state of coma, from which he may again emerge. This sequence may be repeated several times, until death at last takes place. Before this termination there are vomiting, incontinence of urine and fæces, insensibility, and occasionally general convulsions.

In a case reported by Dugast,[1] a woman entered the Hôpital Neckar in a state of marked prostration. Her intelligence was not markedly impaired, but, though she understood almost every thing said to her, she answered only by monosyllables often unintelligible, and pronounced in a low voice. She was affected with paralysis of the left side of the face, and an incomplete paralysis both of motion and sensibility of the right side of the rest of the body.

Four days afterward she was in a state of complete prostration, the paralysis was general. Up to this time the intelligence had remained almost intact. She died that day. The post-mortem examination showed the existence of a large sub-arachnoid extravasation at the base of the brain. On the inferior surface of the pons the blood had become consolidated into a clot which pressed upon the left lobe. On the right side of the pons the blood had not coagulated. This case is interesting as bearing upon the subject of cross-paralysis already considered in the previous chapter.

It has sometimes happened that meningeal hæmorrhage, resulting from an injury of the cranium, has not caused any very prominent symptoms for a considerable period afterward. A teamster was struck on the head by a club in the hands of another man, was stunned for a few minutes, then recovered, and went about his business without complaining of his head. In about twelve hours afterward coma supervened, and he died without being aroused. A case is reported by Dr. Gibson,[1] in which a still longer period intervened. A man, sixty years of age, was found one morning, about eight o'clock, seated as if asleep at a desk, his arms crossed before him, and his head resting on them. It was discovered that he was profoundly insensible. He was sent to the hospital, where he lay comatose, breathing stertorously, and paralyzed on the whole of one side. At the end of two days he died. On post-mortem examination there was found fracture of the left side of the cranium, with rupture of the dura mater and middle meningeal artery, from which latter, extensive hæmorrhage had taken place. It was ascertained that, five days before, he had fallen down a stone staircase, was stunned for a few minutes, but had soon recovered his senses. Doubtless during the whole of the intervening period the bleeding from the ruptured vessel had been going on.

---

[1] "Quelques considérations sur les hémorrhagies méningées cérébrales. Thése de Paris," 1869.

Prus, in the memoir cited, attempts to draw a symptomatological distinction between sub-arachnoideal and intra-arachnoideal hæmorrhage. · Thus for him headache, dryness of the tongue, fever and delirium, are indications of intra-arachnoideal hæmorrhage. Somnolence and coma are common to both forms, but, when they are conjoined with the phenomena mentioned, intra-arachnoideal hæmorrhage is to be diagnosticated. But most authors doubt if the discrimination can in reality be made during life. Valleix[2] declares that the difference is of greater anatomical than symptomatological importance, and Durand-Fardel[3] admits that it is difficult to present a characteristic view of the course and phenomena of sub-arachnoideal hæmorrhage. I must confess that I see no greater anatomical reason for any difference in the symptoms of the two forms of meningeal hæmorrhage than there is for a difference between inflammation of the pia mater and inflammation of the arachnoid. Neither are there any characteristic symptoms which would serve to distinguish hæmorrhage of the dura mater from either of the other forms.

Causes.—Among the *predisposing* causes of meningeal hæmorrhage age occupies the first place. It is more frequently met with in young infants, and in old persons, than in those of middle age. Legendre[4] in two hundred and forty-eight cases occurring in infants, and in which post-mortem examinations were made, found no instance of the child being over three years of age. Between one and two years would appear from his researches to be the period in which children are most liable to the supervention of meningeal hæmorrhage.

---

[1] *Edinburgh Medical Journal*, September, 1870, p. 199.

[2] "Guide de médecine practicien," tome ii., Paris, 1866, p. 4.

[3] *Op cit.*, p. 178.

[4] "Recherches sur quelques maladies de l'enfance." Paris, 1846, p. 113, *et seq.*

But Gintrac's[1] cases are of different import as regards this point, for of one hundred and sixty-five in which the age was noted, only ten were under ten years of age, while thirty-seven were between the ages of thirty and forty, sixty-seven were between fifty-one and eighty years of age, and two of eighty-seven and eighty-eight years respectively.

Meningeal hæmorrhage is often produced by injuries of the skull, and results from sudden rupture of a healthy artery or vein. It may follow blows on the head, falls, or injuries with instruments which perforate the cranium, and may or may not be associated with fractures of the bones.

Extreme heat acting upon the head, venereal excesses, severe muscular efforts, excessive mental exertion, amenrrohœa, overfeeding, and constipation of the bowels, have been cited as exciting causes. The larger vessels, or the capillaries, may give way from being diseased, and consequently unable to resist the ordinary tension of the blood. Such a condition may be the result of the long-continued excessive use of alcoholic liquors, or may be due to hepatic disease.

Prognosis.—The ordinary termination of meningeal hæmorrhage is death. Of thirty-one cases in old persons, cited by Durand-Fardel, death occurred in twenty-six before the end of the fifth day, in one it took place on the seventh day, in two on the fifteenth, and in two in from twenty to twenty-five days. Legendre, in infants, ascertained the duration to be from eight to twelve days. Prus found death to ensue in cases of sub-arachnoideal hæmorrhage before the end of the eighteenth day, but in instances of the intra-arachnoideal form life was sometimes prolonged for over a month.

But recovery has occasionally taken place through the formation of false membranes in such a manner as to circumscribe the extravasation, and thus to conduce to the absorption of its fluid portion, and Legendre has described a process occurring in children by which the sanguineous cyst is transformed into one containing serum, thus producing a species of hydrocephalus. Such terminations are, however, so very rare as to mitigate but to a very slight degree the gravity of the prognosis.

Diagnosis.—The diagnosis of meningeal hæmorrhage is a matter of much difficulty. Still, there are certain characteristics which aid us somewhat in arriving at a correct opinion. Thus, from cerebral hæmorrhage, it may usually be distinguished by the fact that the coma, when it exists, comes on gradually, that the headache is a much more prominent symptom, that there is not often hemiplegia—the paralysis amounting to a general resolution—and above all, by the remissions which so frequently mark its course. Durand-Fardel[2] declares that when the coma and general abolition of the faculties indicate the existence of strong cerebral pressure not accompanied by paralysis, properly so called, or only by incomplete paralysis, perhaps more strongly marked on one side than on the other, we may suspect the presence of meningeal hæmorrhage; that a cerebral hæmorrhage, or an acute softening sufficiently extensive to produce such pronounced symptoms of compression is always accompanied by complete paralysis involving a lateral half of the body, and that the full development of the phenomena is ordinarily preceded by violent headache.

---

[1] *Op. cit.*, p. 738.

[2] *Op. cit.*, p. 168.

From cerebral congestion the diagnosis must be occasionally almost if not altogether impossible, and the same is true of cerebral softening. The remissions when present in meningeal hæmorrhage will afford important assistance in establishing the existence of the disease, but when they are absent the difficulties in the way of an exact discrimination may be insurmountable.

Morbid Anatomy and Pathology.—An extravasation of blood between the cranium and the dura mater, extra-meningeal hæmorrhage, is, as has already been said, almost invariably the result of traumatic cause. Gintrac,[1] however, with his usual industry, has collected five cases in which it appeared to be idiopathic. The first of these he quotes from Dr. J. H. Wythes,[2] of Port Carbon, Pennsylvania, but he omits to state that the child had been playing on the door-step, and that a pain in its ankle was supposed by the parents to have been due to a sprain. It is probable, therefore, that the child fell and struck its head. The next morning it was found dead in bed. On post-mortem examination, an extravasation of blood, amounting to about half an ounce, was found between the skull and dura mater, on the upper surface.

In the other cases the blood appears to have been effused during extreme congestion of the meningeal vessels, one or more of the latter having given way under the excessive tension to which they were subjected. In one quoted from Abercrombie, there were numerous clots scattered over the interior surface of the dura mater, and which seemed to have come from the Pacchionian bodies. These elevations were very vascular, being gorged with blood.

The anatomical characteristics of intra- and sub-arachnoideal hæmorrhages have been very thoroughly given by Prus.[3] In the former the blood is extravasated by exhalation, that is, there is no visible rupture of blood-vessels, and, if life be prolonged to the fourth or fifth day, a false membrane is formed by which the clot is retained in apposition with either the parietal or visceral layer of the arachnoid. This membrane eventually becomes organized by the formation of vessels in it, and may, therefore, be the source of another hæmorrhage; for, as Charcot and Vulpian[4] have shown, these vessels are numerous, large, possessed of very thin walls, and are, therefore, in a favorable condition for giving way under the tension of the blood.

---

[1] *Op. cit.*, tome i., p. 646.

[2] "Three Cases of Infantile Apoplexy." *North American Medico-Chirurgical Review*, January, 1858, p. 70.

[3] *Op. et loc. cit.*

[4] *Gazette hebdomadaire*, 1860, pp. 728, 789, 821.

Brudet[1] previous to Prus had described the false membranes which play so important a part in intra-meningeal hæmorrhage, and had pointed out their resemblance to the arachnoid and their liability to be the source of other hæmorrhages, and at about the period of Prus's publication Mr. Prescott Hewett[2] called attention to extravasations attached to the free surface of the arachnoid, and kept in position by a false membrane not distinguishable by the naked eye from a true serous membrane.

The clot may be extensive, covering nearly the whole surface of a hemisphere. The vessels which have given way, and have thus produced an intra-arachnoideal hæmorrhage, are usually found in an atheromatous condition, and the vessels of the neo-membranes are especially liable to be thus diseased.

Dr. Sutherland[3] in a very interesting memoir gives the details of ten cases of arachnoid cysts occurring in the insane: "On removing the skull-cap and dura mater, instead of the convolutions of the brain, with its vascular pia mater meeting the eye, there appears a reddish, pulpy, fluctuating swelling on the surface of the brain, having such a uniform appearance that the outline of the convolutions beneath it is invisible. On attempting to strip off the cyst from the surface of the brain it is usually found adhering to the visceral arachnoid along the centre of the longitudinal fissure ; it is easily separated from the convolutions on either side ; but if large enough to embrace the entire hemisphere is found again to be adherent below, but in this situation usually to the parietal layer of the arachnoid membrane."

Of the ten cases reported by Dr. Sutherland, four were in all probability due to injury of the head. In five the mental aberration was organic dementia, in three general paralysis, and in two idiocy and imbecility.

In *sub-arachnoideal* hæmorrhages the blood is, as we have seen, extravasated into the space between the arachnoid and the pia mater, and is often entangled in the meshes of this latter membrane. As the blood when extravasated is mingled to a greater or less extent with the cerebro-spinal fluid, it often remains liquid. The quantity thrown out is frequently very large, amounting in some cases to apparently as much as sixteen or even twenty ounces. These figures must, however, be taken with some allowance for the amount of cerebro-spinal fluid with which the blood is combined.

---

[1] "Mémoire sur l'hémorrhagie des méninges," *Journal des connaissances medico-chirurgicales*, 1839.

[2] *Medico-Chirurgical Transactions*, vol. xxviii., 1845.

[3] "Arachnoid Cysts." "West Riding Lunatic Asylum Medical Reports," vol. i., 1871, p. 218.

The anatomical relations are such as to admit of sub-arachnoideal hæmorrhages being very extensively distributed throughout the cranio-vertebral cavity. In one case in which I made a post-mortem examination, it occupied the whole base of the skull, and, in a case cited by Prus, the whole base of the cranium was filled with blood, all the ventricles were in the same condition, and even the sub-arachnoid cavity of the spinal cord was invaded.

New membranes are never met with in this form of meningeal hæmorrhage. Atheroma of the arteries, especially of those at the base of the cranium, is the disease which is ordinarily the immediate cause of the extravasation, and the torn vessel can generally be discovered without difficulty. Aneurisms of the basilar, the internal carotid, or other arteries of the base of the brain, have by their rupture been the cause of sub-arachnoideal hæmorrhage.

Treatment.—There is nothing to add under this head to the remarks already made relative to the management of cases of cerebral hæmorrhage.

PACHYMENINGITIS AND HÆMATOMA OF THE DURA MATER.

A peculiar form of meningeal hæmorrhage, called hæmatoma, is met with under the dura mater. The blood is not diffused, but is collected in sacs which are formed of false membranes, the result of chronic inflammation of the dura mater; or pachymeningitis as it has been designated by Virchow. These capsules are flattened ovals in shape, are three or four inches in diameter, and half an inch thick. They are usually situated at the vertex, and involve both hemispheres. When this is the case, the paralysis which results is bilateral.

Symptoms.—The initial symptoms of hæmatoma of the dura mater are the results of chronic inflammation, and are slow in their progress. In many respects they resemble those indicative of softening, and consist of weakness of intellect, vertigo, a dull, circumscribed, persistent pain, and more or less tendency to stupor. The power of motion is generally diminished on both sides of the body, though occasionally there is hemiparesis. Paralysis is scarcely ever complete. Contractions of the limbs and twitching of the muscles, especially of those of the face, have occasionally been observed. Gradually, through a period extending over several months, the stupor increases, and finally the patient becomes apoplectic. During the whole course of the disease the pupils are strongly contracted. The patient dies comatose and frequently convulsed.

Causes.—Early and old age are both predisposing causes, the disease being met with mainly in children and very old persons. It is frequently seen in the insane, and may probably result from rheumatism, the excessive use of alcoholic liquors, and fevers. The cause is sometimes to be found in wounds or injuries of the skull.

Diagnosis.—It is doubtful if hæmatoma of the dura mater can be definitely recognized either in the stage of inflammation or that of hæmorrhage. Legendre[1] states that, in children, the most important diagnostic mark is the permanent contraction of the hands and feet, which is so generally present; but this symptom is certainly met with in other cerebral disorders, and may even result from reflex irritations. The diagnosis is rendered still more difficult by the fact that the disease under consideration is often associated with other cerebral disorders which mask or modify its symptoms. The absence of fever, the contraction of the pupils, the slowness and irregularity of the pulse, the facts that there are no vomitings and no general convulsions, that the nerves distributed to the several parts of the face are not paralyzed, that there are constant and very severe headache and a gradually increasing tendency to stupor, are, according to Jaccoud,[2] sufficient to indicate the presence of hæmatoma of the dura mater. I am of the opinion that they only enable us to give a guess which has some basis in probability, for I have several times witnessed exactly such a condition as that described, and after death found other morbid conditions than hæmatoma.

Prognosis.—This is unfavorable, death resulting sooner or later, according to the extent of the disease and the natural powers of the patient.

Morbid Anatomy and Pathology.—The first stage of hæmatoma of the dura mater is characterized by the formation of the false membranes, to which allusion has already been made. These membranes are found on the internal surface of the dura mater, and are reticulated, presenting somewhat the appearance of spiders' webs. They generally have their seat near the sagittal suture, and extend to both hemispheres, being only separated from them by the arachnoid and pia mater. Virchow, who has studied their formation with greater care than any other observer, has found more than twenty layers of them, one on top of the other, and traversed by numerous blood-vessels.

Owing to this great vascularity, to the extreme tenuity of the vessels, and to the absence of any perivascular support, hæmorrhage is liable to occur, and the several lamellæ thus constitute a sac into which the blood may be poured. This, pressing upon the cerebrum below, and constantly being enlarged by subsequent hæmorrhages, gives rise to the symptoms observed during life. The vessels may be more liable to rupture from the existence of atheromatous degeneration of their coats.

Anatomically and pathologically hæmatoma of the dura mater differs from intra-arachnoideal hæmorrhage in the facts that the extravasation is between the dura mater and parietal layer of the arachnoid, and that the formation of the membrane precedes the hæmorrhage. Those authors who regard the arachnoid as consisting of but a single layer, and who consequently do not admit the existence of intra-arachnoideal hæmorrhage, must consequently concede that there are two kinds of extra-arachnoideal hæmorrhage, one in which the membrane forms subsequently to the appearance of the extravasation, and the other in which the hæmorrhage is the direct consequence of the formation of the membrane.

---

[1] "Recherches sur quelques maladies de l'enfance," Paris, 1846.

[2] "Traité de pathologie interne," tome i., Paris, 1870.

Others again, as, for example, Gintrac and Durand-Fardel, evidently regard what they describe as intra-arachnoideal hæmorrhage, as identical with hæmatoma of the dura mater; and it is quite certain that many of the cases adduced by Gintrac as examples of intra-arachnoideal hæmorrhage are in reality instances of pachymeningitis with subsequent sanguineous extravasation. The difficulties in the way of a complete understanding of the subject are greatly lessened by remembering the distinction pointed out above, that hæmatoma of the dura mater is a secondary affection, the direct result of inflammation and the formation of false membranes; while in intra-arachnoideal hæmorrhage the membrane is derived from the extravasated blood, which is the first step in the morbid process.

The size of the cysts is subject to much variation, the quantity of blood ranging from one or two to sixteen or even more ounces. By the pressure which they exert upon the brain, the convolutions are flattened, and even softening of the cerebral tissue may be produced.

Treatment.—This requires no amplification at my hands, as I do not believe in the efficacy of any means for curing the affection. All that can be done is to palliate the more violent symptoms, such as the head ache and feebleness of mind and body, by anodynes and stimulants, and of these, morphia administered hypodermically, and alcohol in some one or other of its numerous forms, are to be preferred. Bloodletting and blistering are worse than useless.

CHAPTER V.

PARTIAL CEREBRAL ANÆMIA FROM OBLITERATION OF CEREBRAL BLOOD-VESSELS (ISCHÆMIA).

OBLITERATION of cerebral blood-vessels may take place—

1. By thrombosis of the arteries.
2. By embolism of the arteries.
3. By thrombosis of the veins or sinuses.
4. By embolism or thrombosis of the capillaries.

I.—THROMBOSIS OF CEREBRAL ARTERIES.

By cerebral arterial thrombosis is understood a condition in which an artery of the brain undergoes narrowing of its calibre by the deposition of fibrine from the blood on its internal surface. The clot thus formed is called a thrombus.

Symptoms.—The phenomena observed in consequence of the formation of a thrombus in a cerebral artery are gradual in their development, and are often interrupted by stages of apparent improvement. Headache, as in so many other affections of the brain, is a prominent symptom and is almost constantly present. It is not usually diffused over the whole head, but occupies a place having a close relation in situation with the seat of the disease. It is rarely of a very aggravated characacter, and is remarkable rather for its persistency than its severity. In several cases which have come under my notice, the pupil of the eye of the affected side was dilated from the first, and there were ptosis and strabismus, showing that the third nerve was involved.

At a very early period in the progress of the disease it is not uncommon to meet with marked difficulties in the faculty of speech, and these not only relate to the articulation, but to the memory of words. As regards the first-mentioned form, there may be restraint in the movements of the tongue, the lips, or both, or there may be a loss of coördinating power in the muscles concerned in speech without any actual paralysis. Special inconvenience is, therefore, experienced when attempts are made to pronounce words in which the labial and lingual letters are prominent. The gutturals in such cases are enunciated without difficulty. In the other form in which the memory of words is impaired, the patient is constantly at a loss for language with which to express his ideas; and, though the proper words may be supplied to him, he almost immediately forgets them again. The full consideration of this interesting subject will be found under the head of aphasia.

Vertigo, though generally present, is not usually severe, at least in the early stages.

The incipient symptoms of paralysis soon make their appearance in the majority of cases, and, though there is a gradual advance in the loss of power, there are periods of almost entire remission. Thus the leg, or the arm, or the face, may be the original seat of the paralysis, and eventually the whole of one side be involved. In a case of probable thrombosis in a gentleman now under my charge, the paralysis was at first limited to the muscles supplied by the ulnar nerve and those concerned in deglutition. For one period of five days after I first saw him, there was an entire remission of his symptoms, and he could move his hand and swallow as well as ever, but gradually the power was again lost, and other muscles became involved. At the present time he is almost entirely hemiplegic.

Sensibility is also generally abolished or impaired on the paralyzed side, and thus the various forms of numbness, such as tingling, formication, etc., are present.

The mental symptoms are usually apparent from the first, but may be altogether absent or else so slightly shown as not to attract attention. The memory is impaired, not only as regards words, to which reference has already been made, but also events and circumstances, especially those of recent date. The names of persons and things are likewise readily forgotten. In the case of a gentleman whom I saw in consultation, and in whom I diagnosticated thrombosis, there was left hemiplegia involving both arm and leg, but not the foot, which had begun in the fingers and gradually extended. There was no special difficulty of speech except as regarded the recollection of words, but the memory was wonderfully impaired in every other respect. I entered his room upon one occasion just as the servant was carrying out a tray with the remains of his breakfast. Not three minutes had elapsed since he had eaten, and yet he assured me he had tasted nothing since the day before. The loss of memory was the first symptom observed in this case. Soon afterward he began to improve, and he is now, after fifteen months, free from paralysis, and with his memory almost as good as ever. The loss of memory in such cases seems to be due in the main to the fact that the power of concentrating the attention upon any subject is very much diminished. There is likewise an indisposition to exert the powers of the mind or body, and thus the patient tends to pass into a condition of apathy. Somnolence is a frequent symptom.

An interesting case[1] of what was probably cerebral arterial thrombosis was admitted to the New York State Hospital for Diseases of the Nervous System, August 22, 1870, and came under my observation. The patient, a man forty-one years of age, was temperate, and had never had either syphilis or rheumatism. In March, 1868, he was seized with a dull pain in the right knee, accompanied with numbness. There soon followed formications and pricking sensations, limited to the right foot. These gradually extended upward, and, at the end of two weeks, had reached the shoulder, when he became entirely hemiplegic. During this attack his consciousness was not affected, and his organs of special sense, except his touch, were unimpaired. On the 11th of May following, the patient suddenly lost the power of speech, but experienced no disturbance of consciousness. He remained completely aphasic for four months, being only able during this time to utter a few sounds which could not be interpreted into intelligible words. He then began to enunciate a few words, and gradually acquired more facility, though his power of coördination was far from perfect when he came to the hospital. His paralysis remained complete for nearly a year.

When admitted there was hemiplegia of the right side of the body except the face; his eyesight, hearing, and other special senses, were unimpaired, and his intellect was clear. There was no loss of the memory of words, and no impairment of the motor power of the tongue, but simply a defect in the faculty of coördination of the muscles used in articulation. There was more difficulty in pronouncing labials and linguals than gutturals. Tactile sensibility, electro-muscular sensibility, and contractility, together with the temperature, were markedly diminished in the right arm, while sensibility to pain and deep pressure was normal. The bladder and rectum were not paralyzed.

---

[1] See the author's "Clinical Lectures on Diseases of the Nervous System." New York, 1874, p. 1. Case reported by Dr. T. M. B. Cross.

In talking, he had a peculiar hesitating, stammering manner, highly characteristic of his disease. There were certain words which he was totally unable to pronounce with any degree of accuracy, even after much effort—"Peter Piper"—words which begin with explosive labial letters, and others similarly constructed troubled him greatly. The ophthalmoscope showed the existence of atrophy of both of the disks, and of retinal anæmia.

Under the use of strychnine hypodermically administered, phosphorus, and the primary current to his brain and the faradaic to the paralyzed parts, very marked improvement in all his symptoms was produced. He regained a considerable amount of power in the arm, became able to walk several miles at a time, and acquired the ability to articulate distinctly any words he wished to say. The sensibility returned, and the nutrition of the affected limbs was manifestly improved.

In another case, also the subject of a clinical lecture,[1] there was probably thrombosis of the basilar artery. The patient, a woman, aged thirty-five, while at work wringing out clothes and exerting a good deal of force, experienced a sensation of numbness in the right arm and leg, which was attended with slight loss of power, though not enough to cause her to desist from her labor. At the time of the attack there were no head-symptoms of any kind, and she noticed no paralysis of the face. Her speech was not affected. At the time of her admission into the New York State Hospital for Diseases of the Nervous System, there was paralysis of motion and sensibility of the right arm and of motion on the left side of the face.

The case was therefore one of cross-paralysis, and it was this fact which mainly induced me to locate the lesion in the pons Varolii.

The speech was indistinct, but this was manifestly due to paralysis of the tongue and of the other muscles concerned in articulation.

In the case in question there had been acute articular rheumatism, but the heart was free from functional or organic disease. The attack was not manifested with the suddenness which characterizes embolism, and there were no loss of the faculty of language, and no mental disturbance, which would probably have resulted had the middle cerebral artery been occluded. Besides, the face and the limbs would have been paralyzed on the same side, all of which considerations induced me to believe that the case was one of thrombosis of a limited portion of the basilar artery.

---

[1] *Op. cit.*, p. 130.

During the first stage of thrombosis, before the artery is entirely closed, amendment, and even complete recovery, may take place. The remissions in the symptoms already referred to are due to the establishment of the collateral circulation, and this may become so complete as to eventuate in cure. It must be confessed, however, that the condition of anæmia to which the foregoing symptoms are due, in the great majority of cases ends in softening—a subject which will presently be considered as one of the consequences of thrombosis and other morbid states.

Causes.—Thrombosis of an artery may result from atheroma or from endarteritis, by reason of which its elasticity is diminished and the smoothness of its lining membrane destroyed. Both these conditions retard the course of the blood, and favor the deposition of fibrine on the internal periphery. The walls of the vessels may be healthy, and a thrombus may then be formed through a weak action of the heart—the result of fatty degeneration or other cause impairing its strength.

Certain conditions of the system, such as that which accompanies rheumatism, may induce thrombosis through the excessive amount of fibrine present in the blood and which renders this fluid more readily coagulable. It is probable, also, that other diseases and particular articles of food—as, for instance, alcohol, fat, and starch—when taken in excess, especially when conjoined with insufficient physical exercise, may so alter the composition of the blood—inducing hyperinosis—as to lead to a like result. Inordinate mental exertion, tending as it does to diminish the tone of the arteries by keeping them in a condition of overdistention, may likewise cause the formation of thrombi.

It has apparently resulted from exposure to intense heat, from suppression of the menstrual flow, from severe emotional disturbance, and from blows on the head.

It is much more common in males than in females, and in persons of advanced years than in the young.

Pressure may be exerted upon a cerebral artery by a tumor or other extraneous body, and narrowing of its calibre and a consequent thrombus be produced. Gintrac[1] cites a case of the kind. A young man had suffered for several days with headache and loss of power in the lower extremities. Coma supervened, but he was still able to answer questions. There was then pain in the back of the head, the pupils were dilated, the mouth was drawn to the right, the respiration was laborious but not stertorous, and the left side became completely paralyzed. He died on the fifth day. On post-mortem examination a firm clot was found to occlude the right middle cerebral artery, and it extended to the internal carotid artery, but did not pass into the middle cerebral artery beyond the point of obstruction. At this place in the fissure of Sylvius a small granulated mass, something like a Pacchionian gland, pressed upon the artery and closed it. In such a case the symptoms will of course be developed with much greater rapidity than when the cause of the occlusion resides in the artery itself.

---

[1] *Op. cit.*, tome i., p. 444. Quoted from Roupell, *Medical Times*, 1844, vol. ix., p. 370.

Diagnosis.—Arterial thrombosis is distinguished from cerebral congestion by the facts that the mental and other symptoms are more profound in character, and that the patient has generally passed the prime of life. The existence of paralysis among the early symptoms will likewise tend to the formation of a correct opinion. From cerebral hæmorrhage it is diagnosticated by the circumstance of its gradual development; from encephalitis by the absence of fever and the more chronic nature of the disease; and from embolism by its slow progress and the impossibility of defining the exact period of its beginning.

Prognosis.—The prognosis in cerebral arterial thrombosis is unfavorable, for the reason that, although the morbid process may advance slowly, and may even be spontaneously arrested in its course before the artery is closed, the tendency to complete obliteration is always great, and the chance of sufficient circulation being carried on by the collateral vessels is very remote. The disposition to softening, therefore, always exists, and generally cannot be overcome. The inadequacy of any medical treatment to control the action going on within the artery, or to aid to any great extent in the development of the collateral circulation, is also an element in forming an opinion as to the ultimate result.

Morbid Anatomy and Pathology.—Although Virchow[1] was the first to write distinctly in regard to the nature of thrombosis, the condition was recognized long before his researches were made, and cases of clots plugging up the vessels are to be found detailed by many of the older medical authors, among whom Abercrombie, Carswell, and Cruveilhier, may be mentioned. Since Virchow began his observations in this direction, many instances have been recorded and a large number of memoirs have been issued upon the subject. An interesting case was related by Dr. Packard,[2] of Philadelphia, at a meeting of the Pathological Society of that city held in December, 1859. The patient, who had been under the care of Dr. Heller, was a bachelor, fifty-one years of age. At six o'clock in the morning, at the beginning of February, he was seized with paralysis of the left arm and leg. He was a man of very regular habits, and of fanatical love for every thing instructive, and an accomplished scholar in botany, geography, and languages. The paralysis was soon relieved, and he was able, four weeks afterward, to go out again and to use his arm tolerably well. About the middle of March, in consequence of a fatiguing walk the previous evening, and an attack of diarrhœa during the night, complete paralysis returned. From this he never recovered, but yet did not die till the December following. Previous to this termination he had confusion of ideas and delirium. Upon postmortem examination, among other morbid changes, a cavity in the right corpus striatum was found, and this was surrounded by a spot of softening of the cerebral substance as large as an egg. The basilar artery was completely blocked up with clots, as was also the right carotid. These vessels were atheromatous, and the basilar artery was aneurismally dilated. The clots had all the appearance of being old.

---

[1] Froriep's *Neue Notizen*, 1846, Heft xxxvii.

[2] *North American Medico-Chirurgical Review*, vol. iv., 1860, p. 306.

Dr. Dickinson[1] has brought forward five cases of occlusion of arteries, several of which I am disposed to think were of embolism, instead of thrombosis, as he considers them to be. Dr. Dickinson nowhere alludes to Virchow's investigations, but gives the whole credit of the discovery of the relation between emboli and the formation of concretions in the heart to Dr. Kirkes. The conclusions which he draws from his cases are by no means original, although he evidently so regards them.

The questions to be considered in connection with the morbid anatomy of arterial thrombosis relate to the condition of the artery, the nature of the clot, and the changes which take place in those parts of the brain which are deprived of their due supply of blood.

The affections of the artery, being similar to those which render it liable to rupture, need not be dwelt upon at any length here, as they have already been noticed under the head of the morbid anatomy of cerebral hæmorrhage. Suffice it, therefore, to say that endarteritis and atheromatous degeneration are the diseased states generally met with.

The calibre of the diseased vessel is diminished and the blood is therefore primarily obstructed in its course even before the beginning of the formation of a clot. In addition the internal coat of the artery is roughened, and hence the fibrine of the blood is readily caught and deposited on the internal periphery. Little by little the layer becomes thicker from fresh accretions, until finally the vessel is entirely occluded.

The clot which closes the vessel is, in the beginning, coagulated blood, and hence consists of fibrine and white and red blood-corpuscles. It adheres to the arterial wall and may be of a brown, yellow, gray, or white color. The consistence is greater at the base than at the periphery, and it may contain granules of calcareous matter composed mainly of phosphate of lime.[2] The elements, with the exception of the fibrine, are gradually disintegrated and washed away by the current of blood which continues to flow through the vessel before it is entirely closed, and therefore the layers nearest the arterial wall consist almost entirely of fibrine, and the one nearest the centre of the vessel, which is the latest formed, of fibrine and corpuscles. An examination of such a clot with the microscope shows that the above-mentioned morphological elements are found in its centre, more or less changed, however, according to the age of the formation. A thrombus may undergo purulent softening and disintegration to such an extent as to result in its breaking up into fragments, which may lodge in the vessel or its branches farther on, and thus constitute emboli.

---

[1] "On the Formation of Coagulæ in the Cerebral Arteries." St. George's Hospital Reports, vol. i., 1866, p. 257.

[2] Lancereaux, "De la thrombose et de l'embolie cérebrales. Thèse de Paris," 1862, p. 86.

The region of the brain to which the artery undergoing occlusion is distributed is, of course, deprived to some extent of its blood, and hence presents at first an appearance of anæmia. And this is not prevented by the increase of the collateral circulation, which is never sufficiently vigorous to compensate entirely for the loss by the primary vessel.

Microscopic examination shows the capillaries to be smaller and less numerous than in the normal condition, though there is not any palpable softening.

But after the artery is entirely closed a change ensues. The anæmic portion of the brain becomes red or pink, and this color is deepest on the borders, owing to the collateral circulation which is now fully established. This stage has been called red softening, but I am disposed to think the designation erroneous, and that it is liable to convey false ideas of the pathology. For it is perfectly possible at this time for the anæmic portion of the brain to be restored through the activity of the collateral circulation, with the effect of causing a cessation of the symptoms. If, however, this should be insufficient to provide for the due nutrition of the affected region, softening takes place, and a cure becomes almost impossible.

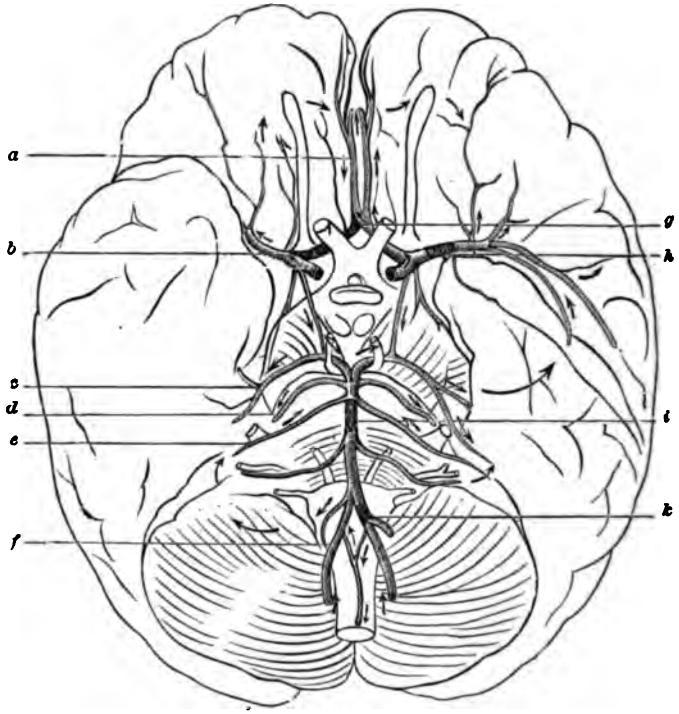
Obliteration of a cerebral artery by thrombus does not always produce notable symptoms. For these to follow, the morbid process must be set up in a vessel with but few and small collateral branches. Thus, if the internal carotid be obstructed, the circulation is carried on through the circle of Willis by the supply of blood derived from the vertebrals. The basilar artery might also be occluded at any limited region between a pair of transverse arteries, and the circulation still kept up by the carotids on the one side, and the vertebrals on the other. But any closure so as to involve one or more of the transverse arteries must lead to anæmia, and subsequent softening of the pons Varolii. Thus, in a case reported by Bennett,[1] in which there had been vertigo and other head-symptoms for several years, and in which paralysis of the left arm, without loss of consciousness, had suddenly supervened, the basilar artery was found entirely obliterated throughout its entire extent, all the transverse arteries were of course closed, and the supply of blood to the pons was cut off on both sides of the mesial line.

---

[1] "Clinical Lectures on the Principles and Practice of Medicine," third edition, Edinburgh, 1850, p. 370.

A somewhat similar case has recently been reported to me by a physician of this city. The patient had suffered with paresis of all the limbs, with pain in the back of the head, occasional vertigo, irregularity of the respiration and circulation, and double facial paralysis for several months. He died suddenly while sitting quietly in his chair. On post-mortem examination the basilar artery was found occluded

Fig. 15.



a, artery of the corpus callosum (anterior cerebral, right); *b*, middle cerebral artery; *c*, posterior cerebral artery; *d*, superior cerebellar artery; *e*, anterior inferior cerebellar artery; *f*, posterior inferior cerebellar artery; *g*, obliteration of artery of corpus callosum (anterior cerebral, right); *h*, obliteration of middle cerebral artery; *i*, obliteration of basilar artery; *k*, obliteration of vertebral artery (left).

and distended by a thrombus which reached from the point of union of the vertebrals to the posterior cerebral arteries, into the left one of which it extended two or three lines.

A very interesting memoir by Hayem[1] alleges occlusion of the basilar artery by thrombus to be a cause of sudden death. In all his cases, four in number, the artery was closed throughout a great part of its extent, as the result of extensive arteritis and the formation of dense clots. In the fourth case there was also thrombosis of the left middle cerebral artery, with difficulty of speech.

---

[1] "Sur la thrombose par artérite du tronc basilaire, comme cause du mort rapide." *Archives de Physiologie Normale et Pathologique*, 1868, p. 270.

The cerebral vessels most liable to be closed by thrombosis are the internal carotid, the middle cerebral, the basilar and the vertebral; after these come the anterior cerebral, the posterior communicating, and the posterior cerebral. It is by no means rare to find two or more arteries simultaneously affected, and in one case cited by Gintrac[1] the whole circle of Willis was obstructed, and, in a remarkably interesting case described by Heubner,[2] the right anterior cerebral artery, the left middle cerebral, the basilar, and the left vertebral were obliterated by thrombosis of syphilitic origin (Fig. 15). The arrows in the figure represent the course which the blood took by reason of the several obstructions to its circulation.

The vessels the closure of which produces the greatest disturbance of function are the anterior, middle, and posterior cerebral, which supply the hemispheres, the corpus striatum, optic thalamus, and other important ganglia. Besides the effect due directly to the anæmia, more or less disturbance results from the congestion posterior to the clot, and the consequent effusion of serum.

Treatment.—A knowledge of the morbid anatomy and pathology of cerebral arterial thrombosis must satisfy us of the insufficiency of any medical treatment to cause the absorption of the clot obliterating the channel of the artery. Yet I have several times heard it gravely proposed to administer the iodide of potassium, with the view of accomplishing this object. As regards facilitating the establishment of the collateral circulation, Nature will generally take care of this, and may even so far overdo it as to cause hæmorrhage from the rupture of vessels not accustomed to the increased tension of the blood. It may therefore be necessary, in this latter condition of excessive action, to give the bromide of potassium in large doses. Should the circulation be feeble, the skin cold, and the patient disposed to somnolence, we have reason to suppose that the collateral circulation is not being formed with sufficient rapidity, and therefore the patient should be kept with the head low, brandy or other spirituous liquors administered, and the body wrapped up in warm blankets.

For some time after the successful establishment of the collateral circulation there is more or less feebleness of mind and body. For this condition strychnia and phosphorus are especially applicable, and may be administered according to the formulas recommended under the heads of cerebral congestion and cerebral hæmorrhage. Electricity is almost always useful.

---

[1] *Op. cit.*, p. 443.

[2] "Die Luetische Erkrankung der Hirnarterien," Leipzig, 1874, pp. 87, 194.

II.—EMBOLISM OF CEREBRAL ARTERIES.

Embolism is the term applied by Virchow to the closure of an artery by an embolus, which is a clot formed in some other part of the body and transported by the current of the blood to the vessel which it occludes. It therefore differs from thrombosis in the facts that it is not associated with previous disease of the artery, and that the closure of the vessel is sudden.

Symptoms.—In cerebral embolism there are no premonitory symptoms. As in cerebral hæmorrhage, the patient may be sitting perfectly quiet when he suddenly loses consciousness and falls to the ground, comatose. As the stupor passes off, he finds that he is paralyzed upon the side of the body opposite to the seat of the lesion.

Or there may be no coma, but merely slight confusion of ideas for a moment or two with sudden accession of paralysis on a limited portion of one side, involving only the arm or leg. Or, again, the face or the tongue may be the only part paralyzed. Or there may be no paralysis anywhere, and no mental symptoms except as regards the faculty of language, which is entirely or partially lost.

Sometimes there are ocular troubles, such as ptosis, strabismus, or blindness.

Experience shows that the embolus, for reasons which will be given hereafter, generally lodges in the left middle cerebral artery, and that with the right hemiplegia—if there be paralysis at all—there is often aberration of the faculty of speech.

The symptoms of mental derangement, with the exception of the coma of severe attacks, are not ordinarily prominent. I have, however, witnessed several cases in which they formed a very striking feature of the case. In one of these, in which the clinical history of the patient disclosed the preëxistence of several attacks of acute articular rheumatism, with subsequent endocarditis and mitral and aortic valvular lesions, there were hallucinations and delusions in addition to the complete paralysis of the left side. All these phenomena entirely disappeared within thirty-six hours. This case is one of the few in my experience in which the embolus had occluded an artery on the right side of the brain.

In another, likewise with valvular disease of the left side of the heart, there was delirium from the first, and this disappeared as the collateral circulation was established.

Erlenmeyer has written very excellently of cerebral embolism, but is, I think, incorrect in some points of his symptomatology. He states the ordinary phenomena of an attack to be as follows:

There are no prodromata; sudden loss of consciousness, with paralysis of several parts of the body. The facial, the hypoglossal, and the nerves of the extremities, are always more or less affected. Sensibility is abolished in the conjunctiva, but is retained in the cornea. The pupils remain sensitive, and are neither contracted nor dilated, neither are there symptoms of concussion or compression. There are no vomitings and no contractions. The pulse is weak and small, and the temperature rather below the normal standard. Occasionally there are epileptiform convulsions. Psychical troubles do not ordinarily appear till the collateral circulation becomes active, and local hyperæmia is thus induced.

The principal exception I have to make to the foregoing sequence of symptoms is the too absolute assertion of the paralysis of the facial, hypoglossal, and other nerves. I have seen several cases in which there was no paralysis to be detected in any part of the body by the most careful examination, and several others are on record. In one very interesting instance, occurring in a lady who had had repeated attacks of acute rheumatism, and who had at the time marked aortic insufficiency, headache and vertigo suddenly occurred while she was conversing with a friend, and her speech was cut short with as much suddenness as though she had been shot. There was no paralysis of the tongue, but all idea of language was abolished. Within forty-eight hours she recovered entirely the faculty of speech. In another, that of a gentleman with a similar clinical history, headache, vertigo, confusion of ideas, and amnesic aphasia, suddenly supervened. That both these were cases of embolism can scarcely, I think, be doubted.

And then, as regards the state of the pupils, my experience does not coincide with that of Erlenmeyer, for I have frequently found either dilatation or contraction of both pupils, or dilatation of one and contraction of the other.

In examining a case of recent embolism, the ophthalmoscope should always be used to view the fundus of the eye, and even in old cases valuable signs will often be obtained. The middle cerebral artery, the ordinary seat of embolus, arises from the internal carotid, after the anterior cerebral and ophthalmic have been given off. Occlusion of its channel must, of course, throw an increased amount of blood into these last-named arteries, and, as the arteria centralis retinæ is derived from the ophthalmic, it and its branches become enlarged. The ophthalmoscope will enable us to discover the congestion thus produced, and will often be the means of helping us to determine, in the absence of paralysis, which side of the brain is the seat of the lesion. In older cases we will frequently find retinal congestion.

The following case I quote not only as being the first of which I have any knowledge in which the ophthalmoscope was used in a case of cerebral embolism, but as being interesting from the fact that the embolus was on the right side. It is reported as

Cerebral Embolism following Valvular Disease of the Heart.—John Turnbull, aged seventeen, was admitted into the Hull General Infirmary, on April 25, 1867. He was tall, much wasted, and had a suffering expression, and converging strabismus of the left eye, the mouth being drawn very slightly toward the left side. Pulse 70, very thrilling in character, and a large coarse systolic murmur near the left nipple. He was perfectly sensible, complained of severe frontal headache, with confusion of vision, and stated that he had been in much the same condition for seven weeks, his illness beginning spontaneously with headache and vomiting, unaccompanied by loss of consciousness or convulsions. He had had an attack of acute rheumatism in the previous summer. He was ordered gr. iij of blue-pill and gr. ij of extract of henbane in a pill, and a draught of acetate of ammonia, three times a day, and spirit-lotion to the head. "No marked alteration in his condition, except progressive debility, took place till May 2d, when he complained of increased headache and dimness of vision, and, being unable to expectorate, from excessive weakness, death from bronchial obstruction threatened. With the aid of some champagne, he rallied in about twenty-four hours, and at the end of a week was much improved, having a clean tongue and good appetite, but the headache, strabismus, and deviation of the tongue to the left, remained. On May 16th it was noticed that these symptoms had passed off, with the exception of the last mentioned. He was ordered a mineral-acid mixture.

"A week later, as he still complained of some dimness of sight, he was examined with the ophthalmoscope. The retinal vessels were found much enlarged, and the veins very tortuous; the optic nerve-entrance of an intense red color, not being distinguishable from the surrounding parts except by the entrance of the vessels, the redness being chiefly due to a number of very fine vessels radiating from the centre. There was no morbid effusion in any part. He could spell easily from No. 15 of Jaeger's test-types (being unable to read and write). He was again examined at the end of another week, when the optic nerve-entrance was observed to be paler in color, so that its circumference could be distinguished, but still much injected, and the vessels nearly as large and tortuous as before; sight was apparently perfect. He was discharged convalescent.

"The peculiar form of paralysis in this case denoted some morbid condition within the cranium, which appeared to have its most easy and natural explanation in cerebral embolism, an opinion further supported by the perfect recovery of the patient. The case received much additional interest from the information afforded by the ophthalmoscope, for one may fairly believe that the intense congestion of the retinæ denoted a similar condition of the brain, perhaps a state of reaction after the circulation had been reëstablished through collateral channels."[1]

---

[1] *British Medical Journal*, 1867; also *Quarterly Journal of Psychological Medicine*, January, 1868, p. 178.

Causes.—The most common first step in the causation of cerebral embolism is acute articular rheumatism, which, by inducing acute endocarditis, leads to the formation of emboli on the valves of the heart and other parts of the endocardium. Aneurisms of the aorta or other large artery, resulting in the coagulation of the blood in the aneurismal sacs, may likewise induce it, by a portion of the clot being washed off by the current. Esmarch[1] details a case in which, while an examination was being made of an aneurism of the carotid, the patient suddenly fell back in an apoplectic stupor. The whole right side was at once paralyzed, the facial muscles on the left side were convulsed, and four days afterward death ensued. Post-mortem examination showed that the left internal carotid, the middle cerebral, and the ophthalmic, were completely closed by coagula, which were identical in structure and appearance with the clot in the aneurismal sac.

Emboli may also originate in the lungs, and, entering the left auricle through the pulmonary veins, finally lodge in a cerebral artery.

Age appears to exercise no influence over the formation of emboli, but men are much more commonly the subjects than women, for the reason, undoubtedly, that they are more liable to attacks of rheumatism.

Of sixty-two cases under my care, either alone or in consultation, in which I had reason to diagnosticate cerebral embolism, there was organic disease of the heart in all but four. Three of the cases were over sixty years of age; seven between fifty and sixty; eleven between forty and fifty; twenty-nine between thirty and forty; and twelve under thirty. Thirty-nine were males and twenty-three were females.

Diagnosis.—From cerebral hæmorrhage, embolism may be distinguished by the following signs. It occurs without relation to age, while hæmorrhage is much more frequent in persons over forty; there are no prodromata; the resultant paralysis is generally on the right side, while in hæmorrhage there is no such predisposition; and it is in the great majority of cases associated with organic disease of the left side of the heart. Care, however, must be taken not to over-estimate the value of this diagnostic mark, valuable as it is. In one case under my charge, in which the symptoms pointed strongly to the existence of a cerebral embolus, and in which, after death, the left middle cerebral artery was found occluded, the heart was perfectly healthy; and in one other, in which cerebral embolus was diagnosticated, and in which there was mitral regurgitation, extravasation into the corpus striatum was discovered to be the cause of death. A case has recently been reported by Dr. J. Hughlings Jackson,[2] in which there was cerebral hæmorrhage with hemiplegia, together with extensive valvular disease of the heart.

A patient now in the New York State Hospital for Diseases of the

---

[1] *Archiv für Pathologie, Anatomie und Physiologie*, B. xi., Heft 5, 1857.

[2] *British Medical Journal*, October 29, 1870, p. 459.

Nervous System has left hemiplegia, involving face, arm, and leg. It has already lasted seven months, although greatly improved. The hand and arm are much contracted. The attack was apparently induced by strong muscular exertion being made while in a stooping and constrained position. Most physicians will be disposed to agree with my diagnosis, that the case is one of cerebral hæmorrhage, for the obvious cause of the paroxysm, the lesion being on the right side of the brain, the steady improvement and the muscular contractions, all point to extravasation of blood instead of embolus. Yet he is under twenty years of age, and, before the seizure, had an attack of acute rheumatism, with heart-symptoms. He now has aortic and mitral regurgitation. Such cases as the above are very instructive, and they show us how necessary it is to weigh all the facts, and how great is the possibility of making a mistake after all. For, although I am inclined to the view of hæmorrhage, no definite opinion can be given without a post-mortem examination.

Still in a case of partial or complete hemiplegia, with or without apoplexy, in which the patient was below the age of forty, with the hemiplegia involving the right side, no muscular contractions and organic disease of the left side of the heart, with or without previous attacks of acute articular rheumatism, cerebral embolus may safely be said to be the cause of the symptoms. Moreover, the paralysis from embolism, if it does not disappear within seventy-two hours after the seizure, does not gradually fade away as it so frequently does to a great extent in hæmorrhage.

It is a somewhat remarkable fact that in cerebral embolism the paralysis may be very extensive and complete without the occurrence of other notable symptoms. Thus in the case of a young lady whom I saw in consultation with Drs. Polk and M. A. Wilson, there had been in childhood a severe attack of inflammatory rheumatism and several minor attacks subsequently. On the last day of September, 1874, she suddenly became hemiplegic on the left side, but did not lose consciousness. There was no aphasia, pain in the head, convulsive movements, nor mental disturbance. The paralysis, however, involved the left arm and leg, and was exceedingly profound. The face was affected for a short time, but the tongue retained its motor power. Three months afterward she could stand and walk a little, but was not able to raise the foot from the ground; the arm was absolutely immovable. Here the clinical history, accompanied as it was with a record of heart-disturbance for several years, was such as to leave no doubt as to the lesion being embolism of an artery—probably the middle cerebral—of the right side of the brain.

The suddenness with which embolism takes place, to say nothing of the other points in the clinical history, will suffice for the discrimination from thrombosis.

Prognosis.—The prognosis in cerebral embolism is grave, for the reason that the tendency to softening of the anæmic cerebral tissue always exists. But, if the patient passes over the first four or five days without any aggravation of his symptoms, and especially if they be mitigated in violence, there is considerable hope of a favorable result. Still, a guarded opinion should always be given till all head-symptoms have disappeared.

Morbid Anatomy and Pathology.—The first rational explanation of embolism was made by Virchow,[1] in 1847, who, in his paper on acute inflammation of the arteries, distinctly explained the manner in which the vessels were occluded by clots transported in the blood from distant parts of the body, and who associated these coagula with valvular disease of the heart. In two of the cases cited by him in which arteries were found closed by such clots, the valves of the heart were discovered to have others still attached to them, and exhibited traces of the separation of those which were found in the vessels.

Subsequently (in 1852), Dr. Senhouse Kirkes[2] called special attention to the plugging up of the middle cerebral artery as a cause of softening of the brain. Three cases, in which death followed, are adduced, in each of which the condition of non-inflammatory softening was found to exist in the brain. Dr. Kirkes's observations appear to have been made without any knowledge of Virchow's prior researches. He states that the paralysis met with in young persons may be due to the interruption of a due supply of nutriment to the brain by the occlusion of an artery by a plug derived from the left side of the heart.

Schützenberger,[3] among others, has written with great fullness on this subject. Among other conclusions not specially applicable to the particular point now under consideration, he states that fibrinous concretions may form in the heart or large vessels, may subsequently be detached and carried by the blood to the cerebral arteries, where they produce symptoms not essentially different from those noticed in cerebral hæmorrhage or acute softening.

The only material points of difference under this head between thrombosis and embolism are, the suddenness of the attack, the part of the brain most liable to be affected, the origin of the clot, and the state of the blood-vessel which is obliterated.

Relative to the first, the abrupt closure of a vessel as in embolism will, of course, produce more violent symptoms than if the occlusion has taken place gradually, and thus time have been afforded for the establishment of the collateral circulation. In the first case, not only is the blood at once shut off from a portion of the brain, but the vessels behind the clot receive a greater quantity than they normally do, and hence the regions they supply are immediately congested. In examination of the brain of a person who has died during the first stage of cerebral embolism, we find those parts of the brain ordinarily supplied by the obliterated vessel paler than natural, with a zone of congested tissue, and perhaps numerous small extravasations of blood on the periphery.

---

[1] "Ueber die akute Entzundung der Arterien." *Archiv für Pathol. Anatomie*, B. i., 1847, p. 272. In a paper on "Occlusion of the Pulmonary Artery," published in Froriep's *Neue Notizen* in 1846, he enunciated a similar theory.

[2] "On some of the Principal Effects resulting from the Detachment of Fibrinous Deposits from the Interior of the Heart, and their Mixture with the Circulating Fluid." *Medico-Chirurgical Transactions*, vol. xxxv., 1852.

[3] *Gazette des Hôpitaux*, No. 80, 1857.

The place where emboli are most frequently found is, as has already been stated, the left middle cerebral artery. The left common carotid arises from the arch of the aorta in a line almost exactly coinciding with the course of the blood-current. It therefore happens that an embolus which has formed on the lining membrane of the heart, and which has passed into the aorta after having been detached, enters this vessel instead of the innominata. From the common carotid it passes into the internal carotid and thence with the stronger and more direct current into the middle cerebral artery, which is lodged in the fissure of Sylvius. Of forty-two cases of cerebral embolism collected by Meissner, in thirty-four the left hemisphere was the seat. Of sixty-two cases occurring in my own practice, and to which reference has been made, fifty were accompanied with right hemiplegia, and were consequently on the left side of the brain. Post-mortem examinations were made in eleven of these cases, and in all the embolus occupied the left middle cerebral artery.

Of these latter was the case of a prominent elderly gentleman of Providence, Rhode Island, whom I was requested to visit in consultation with Drs. Parsons and Collins, of that city. Three days before, while ascending a hill, he had suddenly become semi-unconscious and hemiplegic on the right side. There was also well-marked aphasia. When I saw him he was in a state of partial coma, from which he could be roused so as to be made to comprehend, but was unable to talk, and was entirely paralyzed in the face, arm, and leg, of the right side. The clinical history indicated the existence of disease of the left side of the heart. I diagnosticated an embolus of the left middle cerebral artery, and expressed the opinion that death would ensue within a few hours. In both of these views the other medical gentlemen fully concurred. The patient died about eight hours afterward. The post-mortem examination was made the following day, and proved the correctness of the opinion that had been expressed, for an embolus completely occluded the left middle cerebral artery, at the point where it divides into the branches which supply the island of Reil and the convolutions of the base of the anterior and middle lobes.

The pathology of the genesis of the clot has already been sufficiently dwelt upon in other connections, and the fact that the artery in which it is found is not diseased has been mentioned.

The further consequences of embolism belong to cerebral softening, and will be considered under that head.

Treatment.—It is not necessary to make any remarks on this point in addition to those made in regard to the treatment of thrombosis. There is very little to be done besides meeting indications as they arise, and attempting to relieve the paralysis and other symptoms, for which ends my views have been sufficiently expressed in the preceding chapters.

III.—THROMBOSIS OF CEREBRAL VEINS AND SINUSES.

It was, until the researches of Virchow, generally supposed that the coagulation of the blood in the veins was the immediate result of phlebitis; but through his investigations it is now very well understood that, in the great majority of cases, the inflammation of the veins is a consequence of the formation of a thrombus, and not a cause. For reasons which will be given further on, the sinuses of the dura mater are especially liable to be the seat of autocthonous coagulæ.

Symptoms.—It is very doubtful if venous cerebral thrombosis possesses such a characteristic symptomatology as to admit of its being identified during the life of the patient. There are headache, convulsions epileptiform in character, paralysis of different parts of the body, particularly of the ocular muscles, giving rise to squinting and double vision, disturbances of sensibility, and, toward the close of the disease, coma. Occasionally there is apoplexy at an early stage.

Certain symptoms have been laid down by authors as indicative of the existence of thrombosis of particular sinuses. Jaccoud,[1] however, appears to discredit their importance, and I am disposed to agree with him that, though it may be well to know them, it is safer not to attribute to them an absolute value. Thus, Von Dusch[2] asserts that epistaxis is symptomatic of obliteration of the superior longitudinal sinus; Gerhardt[3] finds a difference in the size of the external jugular veins—that of the affected side being more collapsed than the other—indicative of thrombosis of the lateral sinus; Griesinger[4] states that the presence of a painful circumscribed œdema behind the ear is evidence of the existence of thrombosis of the transverse sinus extending into the veins which lead to the sigmoid fossa; and Corazza[5] thinks obliteration of the superior longitudinal sinus is signified by œdema of the frontal veins, and exophthalmos. An important point in the symptomatology of thrombosis of the encephalic veins and sinuses is the often simultaneous presence of suppurative inflammation of the ear. This is explained by the fact that the relations of the mastoid cells and the petrous portion of the temporal bone to the lateral, the cavernous, and the petrosal sinuses, are so intimate that the extension of a morbid process to them, from the parts of the cranium in question, is readily accomplished.

---

[1] "Traité de pathologie interne," tome premier, Paris, 1870, p. 149.

[2] Henle und Pfeufer's "Zeitschrift für ration. Medicin," B. vii., 1859, p. 161. Also the New Sydenham Translation—"On Thrombosis of the Cerebral Sinuses," London, 1861.

[3] *Deutsche Klinik*, 1857, No. 45.

[4] "Beobachtungen ueber Hirnkrankheiten," *Archiv der Heilkunde*, 1863.

[5] "Revista Clinica," 1866.

Owing to the inflammatory action so frequently set up in the vein or sinus in which a thrombus has been produced, pus enters the general circulation, and hence abscesses are liable to occur in distant parts of the body.

In the very interesting case which forms the basis of Von Dusch's important paper, the principal phenomenon observed during the life of the patient—an infant nine months old—was a large abscess occupying the anterior and outer portion of the right thigh, from which half a pint of pus was obtained, by incision, and which continued to discharge for several days. Death occurred in a few days without being preceded by convulsions, coma, or other head-symptoms. On examination after death, the anterior part of the superior longitudinal sinus was found to be completely closed by a firm, pale, triangular clot of blood, adherent to the walls. Posteriorly the clot did not entirely fill the calibre of the sinus, and was softer. Similar clots were also found in the left lateral sinus, and in the veins terminating in the superior longitudinal sinus.

In a case reported by Abercrombie[1] as "Suppuration within the Left Lateral Sinus," the affection undoubtedly resulted from an extension of inflammation from the cranium to the veins. The patient, a young lady aged sixteen, complained of severe headache, which extended over the whole head. She had an oppressed look, and great heaviness of the eyes; pulse 120; tongue clear and moist; face rather pale. She had been liable to suppuration of the ears, and the left ear had been discharging pus for three weeks; had complained of headache for a fortnight. A few days afterward, her strength began to fail, there was a tendency to stupor, and slight delirium was present. There was constant complaint of pain in the head. Finally, she became more comatose, but was sensible when roused, and knew those about her a few minutes before her death.

On post-mortem examination the membranes of the brain were found congested, but the brain-substance was not diseased. The left lateral sinus was inflamed throughout its whole extent. "Its inner coat was dark-colored, irregular, and fungous. At one place the cavity was nearly obliterated. The disease extended into the torcular Herophili, and affected a little the termination of the longitudinal sinus. Behind the auditory portion of the temporal bone, near the foramen lacerum, and in the course of the left lateral sinus, a portion of the bone nearly the size of a shilling was dark-colored and carious on the inner table. It was at this place that the sinus appeared to be most diseased."

---

[1] "Observations on Chronic Inflammation of the Brain and its Membranes." *Edinburgh Medical and Surgical Journal*, vol. xiv., 1818, p. 288.

It is stated that the walls of the sinus were so thickened as to prevent the passage of the blood, and that evidently no blood had traversed it for some time. Although Abercrombie failed to recognize the real nature of the morbid process, there can be no doubt that the sinus was closed by an old coagulum, which had been adherent to the walls.

Prichard[1] reports the case of a girl sixteen years of age, in whom epileptic convulsions had existed for two years, and recurred very frequently, sometimes several times a day. There appeared to have been no other symptoms indicating cerebral disturbance except that it is mentioned that at one time she was slightly delirious, and then was free from fits. She was treated actively for nine months, and then died in a convulsion, of the usual character.

Examination after death showed that "the left lateral sinus, through its whole extent, was filled up by a substance very different in its nature from a recent coagulum, and apparently consisting of a deposition of lymph, which had become organized. It appeared so completely to occupy the calibre of the sinus as to have entirely impeded the passage of the blood through it."

Another case, reported by the same author,[2] is that of a girl twenty-two years of age, whose mother had been insane, and whose complaint "began by a feverish disorder, under which she labored about nine weeks. It was followed by a melancholy and pensive habit. She was observed to spend most of her time in reading religious books, and attended a meeting of Calvinistic dissenters." When she first came under treatment, her appearance was very wild; she was mischievous, and fond of destroying her clothes. In about three months and a half she was discharged cured, but was readmitted a month afterward, and remained in the hospital till her death, which took place about three years subsequently. During this period her constitutional tendency to scrofula showed itself in a decided manner. The glands of the neck were frequently swollen and inflamed, and she was repeatedly attacked by pneumonic symptoms. When these disorders became a little relieved, her mental alienation was aggravated. She generally sat with her hands folded, and her eyes fixed downward. She died from general debility and exhaustion, but without additional head-symptoms.

Post-mortem examination revealed the existence of thickening of the dura mater, serous effusion between this membrane and the pia mater, fluid within the pia mater, and thickening of this membrane.

---

[1] "A Treatise on Diseases of the Nervous System." London, 1822, p. 176.

[2] *Op. cit.*, p. 357.

The substance of the brain was very firm, the pineal gland was large. "The longitudinal sinus contained a firm coagulum, resembling a polypus, which extended into the lateral sinus."

In only one instance have I had the opportunity of making a post-mortem examination in a case of thrombosis of a cerebral sinus. The patient, a man forty years of age, had been upon a drunken debauch for several days, when he gradually passed into a condition of stupor, which was at first mistaken for the continued effect of alcoholic intoxication. As it continued for two days after all stimulants had been withheld, this idea was abandoned, and the diagnosis of cerebral hæmorrhage was made. I saw him at this time, and was disposed to agree with this opinion. There were profound stupor, stertorous breathing, and complete resolution of all the limbs. Much to my surprise, however, the state of coma gradually passed off, and as sensibility returned the patient complained of intense pain in the forehead and vortex, which was accompanied by twitchings of the muscles of both sides of the face, and of both upper extremities. On the tenth day right hemiplegia suddenly ensued, unattended with loss of consciousness, though there was a slight disposition to stupor manifested as soon as the attention failed to be engaged. The pupil of the left eye was dilated. On the twelfth day a severe epileptiform convulsion ensued, which was succeeded by another on the same day, during which the tongue was very severely bitten. Control of the bladder and rectum was now lost, and on the fourteenth day the convulsive state became permanently established, and the patient died that night without regaining consciousness though the convulsions became somewhat less violent.

The post-mortem examination was made the following morning. The pia mater and arachnoid were somewhat congested, though the subarachnoideal fluid was not notably increased in quantity. The substance of the brain was healthy, and there was no extravasation of blood anywhere to be found. But, on laying open the longitudinal sinus, a firm coagulum was found completely occluding it, from its beginning anteriorly, to its termination in the torcular Herophili, partly filling this cavity, being attached to its anterior wall, and extending for the distance of an inch and a quarter into the left lateral sinus. The thrombus was much more dense and compact in its anterior than in its posterior part, and that portion which occupied the lateral sinus was evidently of more recent formation than the rest.

A consideration of the symptoms exhibited by these cases will serve to show the truth of the assertion made in the beginning of my remarks on the subject, that there are no such characteristic symptoms of thrombosis of the cerebral sinuses as will suffice for the identification of the disease. The most that can be premised is a not very decided probability.

Causes.—Among the causes of thrombosis of the cerebral veins and sinuses, those affections of the heart in which the force of its systole is lessened, and those in which there is an obstacle to the return of the venous blood, occupy a prominent place. Through the action of either of these categories of diseases the circulation within the cranium is retarded, the blood tends to accumulate in the large veins and sinuses, and, its course being abnormally slow, coagulation is liable to ensue. Tumors in the neck, by compressing the internal jugular veins, also tend to the same result by backing up the blood in the lateral sinus. An intra-cranial tumor may exercise a like effect by direct pressure upon a sinus.

Thrombosis may result from the extension of inflammation from the cranium or the cerebral tissue to the sinuses. Such is the case when the suppuration of the ear terminates by the formation of a thrombus in the lateral, cavernous, or petrosal sinus, or when abscess of the brain or an extravasation of blood produces a like effect. The condition in question may also be caused by injuries of the skull; it has been known to follow the operation of trephining and other surgical procedures on the cranium, and may also result from carbuncles, of or near the head, and from erysipelas occurring in like situations.

Age appears to be of some influence as a predisposing cause of venous cerebral thrombosis. Thus, of thirty-seven cases cited by Gintrac,[1] fourteen were between the ages of three weeks and ten years, eleven between eleven and twenty years, six between twenty-one and thirty years, four were forty-five, fifty-five, sixty-five, and sixty-eight years old respectively, and two were of advanced age, not exactly known. As Gintrac remarks, the first period of life is that which is most favorable to the occurrence of venous cerebral thrombosis, adolescence and adult age are a little less favorable, and old age is the least so of all.

Sex seems to be of no predisposing power: of thirty-one cases in which the sex was stated, fifteen were males and sixteen females.

Prognosis.—The elements for forming a prognosis being of a very indeterminate character, it is difficult to form an opinion relative to the probable result in the case of a person presenting the symptoms which have been mentioned. It is perhaps, however, warrantable to say that thrombosis of the cerebral veins or sinuses must from the very nature of the lesion be a most grave disorder, if not one necessarily fatal, sooner or later. If the vein or sinus in which the clot exists be small, and if the causes be of such a character as to admit of removal, and thus the extension of the coagulation be preventable, the prognosis would of course be more favorable than if an opposite state of affairs exists. After all, the only data from which a judgment can be formed are the severity of the symptoms and the course and duration of the disease. The symptoms themselves can be of very little service in this respect, for, as we have seen, they have no such pathognomonic value as to indicate to us the pathological condition with which we have to deal.

---

[1] *Op. et loc. cit.*, p. 528.

Diagnosis.—After the remarks already made incidentally with reference to this point, there is nothing to say which can elucidate the subject.

Morbid Anatomy and Pathology.—The ordinary seat of the affection under consideration, when not the result of some other contiguous lesion, is the superior longitudinal sinus; when due to suppuration of the ear, the clot is usually first found in the lateral sinus; when resulting from injury, it has a near topographical relation to the seat. Through the occlusion of the sinus it becomes distended on the distal side of the clot, and the blood is thus thrown back upon the capillaries and eventually upon the arteries. A state of cerebral ischæmia is therefore induced, to which the symptoms of the first stage of the disease are, in the main, to be ascribed. This ischæmia may lead to extravasation of blood, to inflammation, or to softening. An increased effusion of serum into the sub-arachnoid space and into the ventricles is an almost necessary consequence. The clot differs in character according to its age. When recent, it is soft in consistence and almost black in color, and is not adherent to the walls of the sinus in which it is situated. When old, it is grayish, dense, and unresisting, and attached to the wall of the vessel. If it be divided, a soft, broken-down mass is often found occupying the centre. This consists of fat and other elements of the regressive metamorphosis which the substance of the thrombus has undergone. It was undoubtedly this matter which Abercrombie and other writers mistook for pus.

Other points in the morbid anatomy and pathology of venous cerebral thrombosis have been sufficiently considered in the remarks which have already been made.

Treatment.—There are no means at present known to science by which the affection can be cured, or its consequences prevented. All that can be done is to treat the symptoms as they arise, to search for their cause, and to remove the latter if removal be possible. Life may, in some cases, be prolonged by the judicious use of quinine and stimulants. Convulsions may be lessened in force and frequency by the employment of the bromides, and pain assuaged by hypodermic injections of morphia, by a pill containing half a grain of codeia, given at bedtime, and repeated if necessary, or by directly taking off a part of the intra-cranial vascular tension by leeches to the inside of the nostrils, or cups to the nape of the neck.

IV.—EMBOLISM AND THROMBOSIS OF THE CEREBRAL CAPILLARIES.

The capillaries of the brain may be occluded either by embolism or thrombosis, as are the larger vessels. But the phenomena of these lesions are so indefinite and obscure that it is impossible, in the present state of our knowledge, to identify them during the lifetime of the subject. There is, therefore, little to be said relative to partial cerebral anæmia resulting from obstruction of the blood in the capillaries, other than to call attention to the genesis, the morbid anatomy, and the pathology of the processes in question. It will, accordingly, be more convenient to consider the subject without subdivision into symptoms, causes, etc.

Embolism of the cerebral capillaries may be the result of deposit of *pigment*, of *fat*, of *pus*, or of the *débris of various tissues, normal or abnormal, which have undergone decomposition.*

Pigment may be deposited in the capillaries whenever the blood—as it does in certain diseases—contains an abnormal amount of pigmentary corpuscles. Meckel[1] appears to have been the first to call attention to the condition in question. In the case of a lunatic, he discovered the spleen to be enlarged, and to be covered with dark pigment. Virchow[2] soon afterward, in the case of a patient who had been subject to ague, found the spleen enlarged, black, from excess of pigment, and the blood in the heart to contain cells with pigment. Meckel attributed a great degree of importance to the occurrence of melanæmia—as the blood-disease is called—for the reason that he considered the pigmentary obstruction of the capillaries a condition liable to result therefrom, and, as a consequence, when those of the brain are thus affected, the supervention of head-symptoms. Virchow, however, while admitting the possibility of such a sequence of phenomena, is not able to add any facts tending to elucidate the subject.

Frerichs[3] has called attention to the pigment liver as associated with pigmentary emboli in the capillaries of the brain. Thus he says: "The next organ in point of frequency to the liver, which undergoes important organic and functional derangements, is the brain. Numerous particles of pigment, which have passed unarrested through the vessels of the liver and the lungs, accumulate in the narrow capillaries of this organ, and particularly in those of the cortical substance. Even by simple inspection of the shade of color, we can form an approximate notion of the quantity of coloring-matter which has been deposited, and of the extent of the vascular obstruction. We must not, however, rely entirely upon inspection, for slight accumulations of pigment in the capillaries easily escape notice, particularly when viewed with an unpractised eye, and can only be distinguished with the assistance of the microscope. In addition to the above, it is not at all uncommon for the vessels to become obstructed by a colorless fibrinous-like coagulum which of course does not affect the shade of color. The mechanical interruption to the circulation which is produced in this way, not unfrequently gives rise to rupture of the small vessels, and the formation of numerous capillary apoplexies. Meckel long ago made observations of this nature. Planer described eight cases in which small extravasations were scattered through the gray and white substance of the brain. These numerous hæmorrhages have not come under my own observation; but in two cases I have observed extravasation into the meninges."

---

[1] *Allg. Zeitschrift für Psychiatrie*, 1847, cited by Virchow. "Die Cellular-Pathologie," Berlin, 1871, p. 263, and Jaccoud, *op. cit.*, p. 144.

[2] *Op. cit.*

[3] "Klinik der Leberkrankheiten," Sydenham Society Translation, vol. i., p. 314.

Frerichs states that he has seen three cases in which there were functional derangements indicative of material changes in the cortical substance of the brain. One of them was that of a lady in her fortieth year, who, after an attack of quotidian fever, accompanied by somnolence, suffered from protracted loss of memory. The functions of vegetative life resumed their normal condition, and there were no derangements of motion or sensation present. The headache and giddiness gradually diminished after the removal of the intermittent fever, by means of quinine; but the weakness of memory, and the inability to find suitable words for objects and ideas, were still on the increase two months after the cessation of the ague.

Another case was that of a girl, aged nine years, living in the same district, where, according to the evidence of two medical men, intermittent fever, terminating fatally, was at the time very prevalent. This girl, whose mental powers had previously been normal, had undergone several attacks of tertian fever. After a protracted use of preparations of bark, she recovered in her bodily symptoms; but her mental faculties gave way, and a state of complete idiocy, accompanied by a ravenous appetite, supervened.

In regard to these cases, Frerichs further remarks that it is uncertain whether atrophy of the brain had resulted from occlusion of the capillaries, or whether it had been induced by the extensive capillary apoplexies consequent upon this occlusion, or whether the intermittent fever was complicated with other accidental changes in the brain. He gives the details of several other cases of intermittent fever, accompanied by head-symptoms, and in which, after death, the cerebral capillaries—principally those of the cortical substance—were occluded by deposits of pigment, originating in the liver and spleen, and transported to the brain by the current of the circulation.

A case is reported by Bright,[1] of a man, who died of paralysis following fever, in whom the cortical substance of the brain was the color of black-lead.

Sydenham had not failed to notice the fact that mental derangement sometimes remains after intermittent fever, which, if treated by depletion, passed into imbecility.

Cases of like character have frequently come under my notice. In one of these there had been repeated attacks of intermittent fever, and the spleen was greatly enlarged. The patient, a young man twenty-two years of age, had suffered from epilepsy for several months, the first paroxysm ensuing shortly after a severe seizure of fever, and being preceded by headache, vertigo, confusion of ideas, and twitching of the muscles of the face. When I first saw him his mind was considerably impaired, and he was having three and sometimes four or five epileptic fits every week. All his mental symptoms were improved by the use of arsenic; his fits ceased, and his spleen became much reduced in size.

---

[1] "Reports of Medical Cases," London, 1801, chapter ci., plates xvii. and xix.

Those physicians who have practised in malarious regions can scarcely have failed to notice the fact that the enlarged livers and spleens, which are so frequently produced by repeated febrile attacks, are often coexistent with cerebral symptoms, such as have been described.[1]

The vessels of the cortical substance appear to be more liable to occlusion from pigmentary emboli than any other part of the brain. Some recent researches of my own would seem to show that the vessels of the retina are also apt to be so obstructed, and that some cases of pigmentary deposit in the eye are in reality instances of pigmentary embolism of the intra-ocular vessels.

Although the *symptoms* of the affection in question have nothing characteristic about them, yet its existence may be suspected with some show of probability, when pain in the head, delirium, convulsions, vertigo, paralysis, and other disturbances of sensibility and motility, coexist with enlarged spleen or liver, and when there is the previous history of malarial fever.

Embolism of the cerebral capillaries from migration of *fat* is a condition which certainly occurs, but which has not as yet been very thoroughly studied. Todd,[2] in a woman who died comatose and hemiplegic, found after death an extravasation of blood into the right corpus striatum, and that "the vessels of the softened portion of the corpus striatum, immediately surrounding the clot, were thickly studded with oil-globules, which in some situations were aggregated into dark masses so large as here and there almost to fill up the vessels. The minutest capillaries, as well as the larger arteries, exhibited these deposits, and few could be discovered without them.

Bergmann,[3] who has devoted much attention to the subject of fat embolism, has recently[4] reported a case in which a man, who died in consequence of injuries received from a fall, was found to have many hæmorrhagic extravasations into the lungs, and numerous oil-globules in the pulmonary capillaries. The brain does not appear to have been examined, but probably the cerebral capillaries would have been found in a like condition.

---

[1] A further consideration will be given to this very interesting subject in the forthcoming memoir of the author, on "Pigmentary Cerebral Embolism, and other Affections of the Nervous System the Results of Malarial Poisoning."

[2] "Clinical Lectures," London, 1861, p. 733.

[3] "Zur Lehre von der Fettembolie." Dorpat, 1863.

[4] "Ein Fall tödlicher Fettembolie." *Berliner klinische Wochenschrift*, No. 33, 1873.

In order to throw additional light on this subject, I have performed a number of experiments upon animals, of which the description of one will be sufficient, as the results were analogous in all essential respects.

Into the left ventricle of the heart of a medium-sized dog sixty minims of olive oil were injected.[1] The animal was killed six hours afterward by section of the medulla oblongata. The brain was removed from the skull and carefully examined. The membranes were decidedly congested. The arteries of the base of the brain contained numerous oil-globules, and this was especially the case with both the middle cerebral arteries. The minute terminal branches of these vessels were filled with fat, and several of them were entirely occluded. The microscope showed the capillaries throughout the brain, both of the cortical and medullary substance, to be gorged with fat-globules, aggregated in masses, so as to prevent, in many instances, the passage of the blood.

Fig. 16

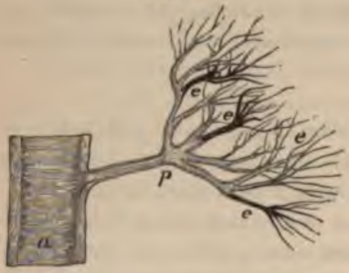
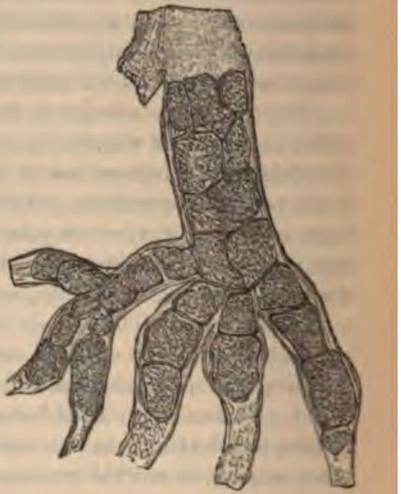


Fig. 17.



In other experiments I allowed a longer time to elapse before killing the animals, and in one death took place spontaneously during a state of profound coma. The post-mortem appearances were more strongly marked, and in the latter several centres of incipient softening had been set up.

---

[1] The heart was penetrated through the thoracic wall by the needle of an hypodermic syringe, and the injection made very slowly. The left ventricle was chosen in order to avoid, as far as possible, the stoppage of the oil in the lungs.

Nothing is known relative to the symptomatology or pathology of fat-embolism of the cerebral capillaries, or of the elements of a correct diagnosis or prognosis of the affection.

The cerebral capillaries may be obliterated, as Virchow[1] has shown, by deposits of *pus* or of the *débris of organic structures* undergoing disintegration. Thus a thrombus undergoes such a transformation that a puriform mass originates in its centre through changes taking place in the central layers of the clot, and the whole eventually becomes converted into a finely-granular substance which is capable of being transported to distant parts of the body and occluding the smaller vessels and the capillaries; or, for instance, ulceration following endocarditis takes place in one of the cardiac valves, as a consequence of acute or chronic softening. The minute fragments of the valve are carried away by the current of the blood, and are deposited in the vessels of remote parts, such as the eyes, the brain, the kidney, and spleen. The accompanying cuts (Figs. 16 and 17) represent these capillary emboli in the penicillii of the splenic artery, following endocarditis. In Fig. 16 the vessels are magnified ten diameters; in Fig. 17 three hundred.

Whether such emboli are capable or not of transferring specific disease to other parts where they are deposited, or whether, as some authors, differing from Virchow, assert, they merely act in a mechanical manner, is as yet undetermined. The weight of evidence appears to favor the view of Virchow, that they act not only by occluding the capillaries, but also by their inherent specificity originating new centres of local disease.

THROMBOSIS.—Thrombosis of the cerebral capillaries may, like the same condition of the larger vessels, result from any cause capable of inducing a stoppage or retardation in them of the circulation of the blood. One of the most common of these factors is calcareous deposit, a state which is only to be detected after death, and which, like many other analogous morbid processes, was first clearly pointed out by Virchow.[2] According to him it depends upon the failure of the kidneys to excrete the mineral matter which is taken up by the blood from the bones, and which in consequence is deposited in other organs.

Some authors regard calcareous deposit as being a process more analogous to embolism than to thrombosis, but it must be recollected that the mineral substance is not in a morphological state in the blood, but is held in solution up to the time of its separation at the places where it is found. It would, in my opinion, be equally logical to regard the deposition of fibrine upon the internal coat of a vessel as embolism, for it is held in solution till it becomes attached to the wall, and in this respect does not differ from the condition of the calcareous matter.

---

[1] "Die Cellular-Pathologie," Berlin, 1871, p. 237, *et seq.*

[2] *Op. cit.*, p. 252.

In the first place, the serum of the blood holding the mineral substance in solution is probably infiltrated through the vascular walls into the peri-vascular tissue and the deposition effected there. Eventually, as the change in the surrounding substance tends to prevent further transudation, and as the vessels degenerate from their normal structure, the metastatic deposit is made around their internal circumference and the channel is finally occluded. At the same time the capillaries lose their elasticity and become hard and brittle. The brain in the vicinity of these centres of morbid action may be so saturated with the calcareous matter as to give a distinct grating sound when cut, and the molecules of phosphate or carbonate of lime may even be seen with the naked eye and distinctly felt when a portion of the brain is rubbed between the fingers.

Marcé[1] reports the case of a man, fifty-five years of age, who died in a state of complete dementia. On post-mortem examination the membranes were found adherent to the brain; in the centrum ovale of both sides there existed large lacunæ of a yellow color and with the appearance of elder-pith. In addition, there were numerous calcareous incrustations forming sharp protuberances and giving a sensation to the finger like that experienced when the tongue of a cat is gently rubbed. The capillaries were likewise incrusted. The cerebral substance contained several old hæmorrhagic *foyers*. The calcareous concretions were found to consist of crystallized carbonate of lime and of the same substance in globular masses. Subjected to the action of dilute hydrochloric acid, they were dissolved with the evolution of carbonic-acid gas; an organic substance analogous in its characteristics to the corpora amylacea remained; it was not, however, colored blue by iodine.

The capillaries surrounding these masses had undergone various degrees of calcareous incrustation. On some, the crystals were scattered here and there on the walls; on others they formed groups or *plaques*, more or less enveloping the circumference of the vessel. There were some in which the channel was entirely obstructed by the colorless crystals, without any other foreign matter, fatty, granular, or pigmentary, being present.

Thrombosis of the cerebral capillaries may also be the consequence of atheromatous degeneration and of moniliform dilatation.

The white substance of the cerebrum, the cortical layer, and the corpora striata are more liable to be the seats of this process than the other parts of the encephalic mass.

---

[1] "Bulletin de la société anatomique," 1863, p. 468, cited by Gintrac, *op. cit.*, p. 473

CHAPTER VI.

CEREBRAL SOFTENING.

As a consequence of several of the conditions described in the foregoing pages, and especially as resulting from thrombosis and embolism in their various forms, cerebral softening naturally comes next in order for consideration. Most authors treat of it in direct connection with obliteration of the cerebral arteries; but, although frequently due to this cause, it may be produced by others, and occlusion is not always followed by softening. For these reasons I have preferred to consider it as it really is, a distinct pathological condition—as much so as sclerosis or any other morbid anatomical state.

Symptoms.—When softening is the result of hæmorrhage, of arterial embolism, or of arterial or venous thrombosis or embolism, the symptoms peculiar to those affections are first met with. Thus there are troubles of the intelligence, the sensibility, and the power of motion, such as have already been described under the heads mentioned, and, if the morbid process goes on within the cranium to its full development, there are peculiar aggravations and the evolution of new symptoms. If coma has existed from the beginning, it may continue with little or no remission, and the patient may die without regaining consciousness, or may become only partially sensible. The condition of softening is not usually set up after either hæmorrhage, thrombosis, or embolism, till about the tenth day, though some cases are more rapid in their progress, and the symptoms now to be mentioned are those which are coincident with what some pathologists have designated the "second stage;" the "yellow softening" of others. The "first stage," or "red softening" of these writers, is, in my opinion, not in reality softening, but rather the congestion due to overaction in the collateral circulation.

In addition to the continued paralysis of motion and the loss of sensibility which exist on one side of the body, the mental symptoms become more strongly marked. There may be delirium with the occurrence of hallucinations and delusions, though these are generally evanescent. Occasionally a fixed idea obtains possession of the patient's mind, and for a while influences him in his conduct, but his mental tenacity is not strong enough to enable him to retain it for any length of time, so it soon yields to another.

The intelligence is notably diminished, so that the patient is unable to conceive an exact idea of his situation, or to obtain a moderately complete notion of quite simple matters which may be submitted for his mental action. Thus he refuses to credit the assertion that he is ill, declares that his health, both in mind and body, is excellent, and that he is fully capable of transacting his business or of performing any intellectual operation.

The memory is invariably impaired, and things of the greatest familiarity are forgotten. Thus a patient laboring under cerebral softening, the result of embolism, could not tell his wife's name, nor by what means he came to my office. Another, sent to me by Dr. Michel, of St. Louis, in whom thrombosis was the probable cause, could not tell me where he came from, nor the names of his children. He insisted with great vehemence that he was perfectly able to attend to his ordinary business, and yet was unable to add three numerals together.

In another case, likewise having the clinical history of thrombosis, which I saw in consultation with my friend Dr. J. W. Ranney, of this city, the patient, a gentleman of about sixty years old, could not tell his age; declared that Dr. Ranney, whom he had known for many years, was a grocer, "who lived around the corner;" and held to the delusion that his sons had made several forcible attempts to rob him.

The power of giving the attention to subjects is very greatly lessened. The patient may seem to be listening to what is said, or observing what is passing about him, but, if he be questioned he at once shows that he really has not been heeding; even when things are forcibly brought to his mind, and he is told to mark them, he is incapable of doing so to any considerable extent.

The speech is almost invariably affected either in the form constituting aphasia, or from paralysis of the tongue and other muscles concerned in articulation. There is a disposition to misplace words, or to clip them by cutting off the last syllable. Thus a patient reading the title of a book in my library called it the "Unit. Stat. Dispenst." for United States Dispensatory; another was the "Philosoph. as Absol. Scien." for Philosophy as Absolute Science; and he told me he was "a lawy. by professi.," when he meant to say he was a lawyer by profession. The same fault is shown in reading from a printed page, and in writing. Only a few days ago I received a letter from a gentleman, in which the final letter of nearly every word was omitted. The emotions, especially those of a sorrowful character, are very easily excited, and therefore the least untoward event causes the exhibition of feeling. Sometimes the patient sheds tears without being able to assign any cause, or may get into uncontrollable fits of weeping; occasionally of laughing.

All these symptoms indicate failure of the mental power, but it is, nevertheless, true that softening of the cerebral tissue may exist without the manifestation of the least degree of imbecility. It not unfrequently happens that, while there is a general loss of intelligence, some one or two faculties of the mind are notably increased in vigor.

I have a patient now under my charge whose intellectual force *is* greatly reduced, who cannot pronounce the simplest sentence *correctly,* who is paralyzed throughout the whole of one side, and who has so lost the sense of propriety that if he feels the desire to urinate he yields to it at once, no matter where he may be or who are present, but whose volitional power is even greater than before the accession of his disease. Thus he will read volume after volume, turning over the pages regularly, and scarcely, except by oversight, skipping a word, although it is very certain he does not comprehend a tenth part of what he reads, and that what he does for a moment understand is immediately forgotten. The strength of his will is also shown in the impossibility of inducing him to do any thing which either caprice or habit prompts him not to do. His appreciation of harmony has become so sensitive that a discord of sounds made on the piano causes him real mental suffering, whereas when he was in health his musical taste and discrimination of the pitch and quality of sounds were below mediocrity.

Drowsiness is very generally present; at first, perhaps, to a slight extent, but sooner or later as a prominent feature. Headache is very common, and is usually dull and circumscribed. The forehead is its most common seat. Other sensations in the head, such as vertigo, fullness, weight, and constriction, are scarcely ever absent.

Gradually, the condition of the patient, mentally and physically, becomes weaker and weaker, and death ensues, immediately preceded by coma, convulsions, delirium, or a combination of these phenomena.

Not unfrequently, softening of the brain is not preceded by hæmorrhage, thrombosis, embolism, or other evident affection, but begins obscurely, and advances very gradually. Such cases are often directly due to disease and obliteration of the cerebral capillaries, as described in the immediately preceding chapter, or they may be the result of a slow inflammatory process. In this form the symptoms make their appearance in succession; but the paralysis, instead of being present from the inception, comes on very slowly, commencing as a slight weakness, conjoined with numbness, in one or more of the extremities, or in the face. Ordinarily, the first evidence of paresis is discovered in the leg, which is not lifted clear of the ground. The toe consequently strikes against the inequalities of the pavement, and the patient is apt to fall. Sometimes the weakness is shown by the leg suddenly giving way at the knee. I have had several patients with cerebral softening, in whom this accident was of common occurrence, and who had thereby received severe injuries. Or, when the arm is the paretic member, the grasp, as shown by the dynamometer, is materially lessened in strength, and things held in the hand are dropped. I have now a patient in charge in whom the affection is in its very earliest stages, and of which the only manifestations are, clipping of the words in speech and paresis of one arm.

This inability of the muscles to maintain a continuous contraction for a short time, though met with in several other affections, is to some extent characteristic of cerebral softening, and, in conjunction with the other phenomena, is a valuable indication. Even before it has become so far developed as to attract the attention of the patient or those about him, its existence may be ascertained by means of the dynamograph described in the preliminary chapter of this treatise. It will often be found that a straight line cannot be made, but that the pencil pursues a zigzag course, or else one descending with more or less regularity.

The paralysis usually goes on to complete loss of power, though its progress is often very slow, and is marked occasionally by periods of decided improvement. At these times the patient's friends imagine that he is about to recover, and if, as is sometimes the case, the mental symptoms are likewise mitigated, their hopes are still further exalted. It is necessary that the physician should not be deceived. In a case which I saw in consultation with Dr. Chamberlain, of this city, I diagnosticated chronic softening. At the time, there were feebleness of memory, paresis of one side of the body, and difficulties of speech. I gave an unfavorable prognosis, but soon afterward amendment began, and the patient, who was an insurance agent or appraiser, resumed his business to some extent. I nevertheless adhered to my opinion, for I had seen too many cases of similar character to be deceived in so clear a one as this. I never saw the patient again, and am therefore unacquainted with the subsequent phenomena, except that about a year afterward I was invited by Dr. Chamberlain to be present at the post-mortem examination. His brain contained a *foyer* of softened tissue as large as a walnut, apparently the result of obliteration of the posterior branch of the left middle cerebral artery, and involving a portion of the middle lobe of the left hemisphere.

In another case, which I had very thorough opportunity for studying, the patient, a gentleman thirty-five years of age, was the subject of chronic softening, without any history of previous lesions. The disease had come on very insidiously, first showing itself by a slight impediment of speech and impairment of memory. Gradually he lost power in both arms and both legs, though the right side was more affected than the left. His gait became titubating, and although he never lost the ability to walk, yet he did so with great and increasing difficulty. But his stages of apparent improvement were at first numerous and well marked. His memory at such times was stronger, his countenance brighter, his articulation distinct, his emotions more under command, his power of attention increased, his intelligence equal to all ordinary occasions, and his walk free from any sign of debility. Then all these steps would be suddenly lost, and he would again become imbecile and weak. Finally, a severe convulsion, more evident on the right side than the left, supervened one evening after dinner, as he was quietly smoking a cigar. Between seven and twelve o'clock that night he had over a hundred fits. He died at the latter hour. The post-mortem examination revealed the existence of a large centre of softening, involving the middle lobe of the left hemisphere.

Sometimes the course of the disease is still more irregular. No evidence of cerebral disorder is perceived beyond aphasia, and the patient remains in the full possession of his intellect, and without paralysis, up to a short time before death. Durand-Fardel[1] cites the case of a man, thirty years of age, who entered the Hôtel Dieu, presenting all the signs of pulmonary phthisis. In a few days afterward he experienced difficulty of articulation, in thirty hours he became comatose, and, in twenty more, died. The post-mortem examination revealed the existence of softening of the inferior surface of the left middle lobe of the cerebrum. Although it is not so stated—Durand-Fardel having written previous to Virchow's observations—there is little doubt that the cause of the softening was an old embolus in the left middle cerebral artery.

Lallemand,[2] in his first letter, cites several cases in which the disease was marked by singular symptoms, such as convulsions, contractions, and delirium.

In a case which I saw in consultation with Prof. C. A. Budd and Dr. J. T. Taylor, occurring in a gentleman about thirty-five years of age, there were coma and violent hemi-convulsions, evidently due to softening from embolism, of which there had been two attacks, the last several weeks previously. Death ensued, but no post-mortem examination was, I believe, obtained.

A gentleman is now under my charge who has valvular disease on the left side of the heart, the consequence of rheumatic endocarditis, and who, six months since, had an apoplectic attack conjoined with aphasia and right hemiplegia. He soon became able to speak pretty well, and regained power and sensibility to a great extent in the paralyzed limbs. During the past two weeks, however, he has exhibited symptoms of mental derangement, as shown by the existence of hallucinations and delusions, and is gradually losing the power of motion and of sensation on the right side. His speech is as perfect as it ever was, and there is yet no sign of dementia.

It has happened that individuals have died who, on post-mortem examination, were found to have softening of the brain, but who, during life, had exhibited no symptoms of this or any other cerebral disorder. Rostan, who was the first to write systematically on the disease, refers to such cases, and Durand-Fardel is still more explicit. The latter says:

"We meet with softening of the brain in persons who, up to the time of death, had presented no appreciable derangement of the cerebral functions, and in whom softening has been developed without having given any evidence whatever of its existence." In such instances the white matter of the hemisphere can alone be involved.

---

[1] "Traité du ramollissement cérébrale," Paris, 1843.

[2] "Recherches anatomico-pathologiques sur l'encéphale et ses dépendances," Paris, 1824.

One such case verified by post-morten examination has occurred within my own experience. The patient, a soldier of the Second United States Infantry, died at Fort Riley, in Kansas, of which post I was medical officer, of chronic dysentery, the result of exposure. There were no mental symptoms, no difficulty of speech, no paralysis; nothing, in fact, indicating the existence of brain-disease. He died in full possession of his intellectual faculties. The post-mortem examination revealed the existence of ulceration of the small intestines, and, as the cause of death was very evident, the brain was not examined. I reserved it, however, for purposes of study, and, on making a section of the right hemisphere an hour afterward, discovered an encysted centre of softening, including more than two-thirds of the posterior lobe. The right posterior cerebral artery was entirely obliterated by thrombosis. The man had been at the fort several months, and had never made complaint of any illness till he was attacked with dysentery six weeks before.

The duration of cerebral softening is very variable. Rostan found it to range from a few days to several years. Andral, from an analysis of one hundred and five cases, found that the period was from twelve days to three years. The most rapid case occurring in my experience terminated in death at the end of eighty hours. Some confusion on this point has arisen from the fact that some authors regard embolism and thrombosis as essentially identical with softening, a doctrine which is clearly erroneous, as, in many cases of these affections, recovery or death may take place without the stage of softening being reached. In the case above referred to, post-mortem examination showed that the condition known as yellow softening was just making its appearance. As I have already stated, I cannot regard the alteration called by some pathologists red softening any thing more than the congestion due to the active collateral circulation.

The case of longest duration, of which I have any personal knowledge, was that of an eminent scientific gentleman, who had suffered from the symptoms of softening of the brain for nearly four years, when he died. There was no post-mortem examination, but the history of the case was that of thrombosis of the left middle cerebral artery, and the course of the disease left no room for doubt as to its nature.

The symptoms of cerebral softening which I have specified are those which are in general the result of the morbid processes existing in the cortical substance of the hemispheres, or in the optic thalami, or corpora striata. Generally, as Laborde[1] has shown, whenever the cortical substance is the seat of softening there is at least one other centre occupying the central part of the brain, or especially the corpus striatum or optic thalamus. But the other portions of the encephalic mass are liable to be similarly affected, and then the phenomena are of a different character.

---

[1] "Le ramollissement et la congestion du cerveau principalement considérés chez le vieillard," Paris, 1866, p. 1, *et seq.*

Thus the pons Varolii may undergo softening from occlusion of the basilar artery, or of one or more of its transverse branches, or from disease of its capillaries, or from chronic inflammation of its substance, and if the disease be limited to this ganglion there is no marked mental deterioration or other evidence of intellectual derangement. The symptoms are in the main connected with sensibility, and the power of motion with articulation, and with the respiratory, circulatory, and stomachal functions, as evidenced by dyspnœa, irregular action of the heart, and nausea and vomiting. In the case of an elderly gentleman whom I saw in the early part of 1874, and who had been affected for about a year, there was almost complete paralysis of the lower part of the face on both sides, there was great difficulty of swallowing, the tongue could not be protruded, speech was very indistinct, the respiration and action of the heart were irregular, and the limbs were partially paralyzed. There was a general loss of sensibility throughout the whole body, and attacks of vertigo and epileptiform convulsions had been frequent. At the same time the intellect was as clear and exact in its operations as it ever had been. I diagnosticated glosso-labio-laryngeal paralysis, and expressed the opinion that the patient would not live over a month. He died in two weeks. The post-mortem examination showed the hemispheres and cerebellum and the membranes to be healthy. The basilar artery was entirely closed by a thrombus. The pons Varolii was as soft as cream, and the membranes peeled off as easily as if they had never been attached to it. Examined microscopically after due preparation, the capillaries were found to be in a state of atheromatous degeneration. The medulla oblongata was not softened, but extreme atrophy of nerve-cells had taken place in the nuclei of the facial nerve of both sides. This point will be further considered under the head of atrophy of nerve-cells.

Softening of the cerebellum can scarcely, in the present state of our knowledge, be diagnosticated from any other affection of that organ. The rapid form, such as results from embolism of the larger vessels, presents so many analogies with hæmorrhage that there are no sure signs by which a discrimination can be made; and the slow form due to disease of the capillaries or to chronic inflammation is not distinguished from abscess or tumor. But it may be inferred that the cerebellum is the seat of structural change when the category of symptoms cited under the head of cerebral hæmorrhage is present, and the history of the case will often aid us in forming an opinion of its nature not very wide of the mark.

When death results from cerebral softening, it may be directly due either to the disease itself, or to some intercurrent affection. Thus the patient may die from pure exhaustion or from slow asphyxia caused by the imperfect action of the respiratory function, or he may choke to death either by being unable to swallow food which he has taken into his mouth, or by the regurgitation of the contents of the stomach during a convulsion, or a severe convulsive seizure may cause immediate asphyxia, or a series of convulsions may produce a more gradual asphyxia, or he may die in a state of profound coma.

The intercurrent affections may be either meningitis or hypostatic congestion of the lungs from long confinement to the recumbent posture, or diarrhœa, or a fresh attack of thrombosis or embolism.

Causes.—The etiology of cerebral softening has already been considered to some extent under the heads of cerebral hæmorrhage, and obliteration of cerebral arteries and veins and of the capillaries, from embolism and thrombosis, of which conditions it is so often a sequence; but, as it may occur without having been preceded by either of these or other noticeable affections, a few additional observations are necessary.

Age is certainly a strong predisposing, if not an actual exciting cause, although the disease is observed at all periods of life. Rostan, whose cases were collected at the Salpêtrière, a hospital containing only old women, found that there were ten cases in persons between the ages of sixty and sixty-nine, twenty between seventy and seventy-nine, and ten between eighty and eighty-seven. Andral, excluding cases occurring in infants, found that, of one hundred and fifty-three cases, there were between the ages of

15 and 20	10
20 " 30	18
30 " 40	11
40 " 50	19
50 " 60	27
60 " 70	34
70 " 80	30
80 " 89	4

Durand-Fardel, from an analysis of fifty-five cases, found between the ages of

30 and 40	3
40 " 50	8
50 " 55	2
60 " 70	14
70 " 80	23
80 " 87	5

The period of life, therefore, at which softening is most apt to occur, is from the age of fifty to eighty.

During the past ten years, forty-five cases of cerebral softening, not the result either of hæmorrhage, arterial embolism, or of arterial or venous thrombosis, have been under my care or been seen by me in consultation. Of these, one was under twenty years of age; four were between twenty and thirty years; nine between thirty and forty; twelve between forty and fifty; eight between fifty and sixty; eight between sixty and seventy; and three between seventy and eighty. The general results, therefore, go to show the greater proclivity which advanced age gives to the occurrence of the disease. In one of those between seventy and eighty, the mind was scarcely impaired till about two months before death, though there had been paresis, headache, and aphasia, for two years.

No definite statistics have been collected relative to the influence of sex, although the opinion appears to prevail that the affection is more liable to occur in females than in males. Of the forty-five cases just cited, twenty-nine were males and sixteen females.

The season of the year does not appear to exercise much influence. Durand-Fardel, from sixty-three cases, found that seventeen occurred in winter, thirteen in spring, twenty in summer, and thirteen in autumn. I have found it difficult in many cases, from the insidious or latent character of the early symptoms, to fix the period of beginning with accuracy.

Intense and long-continued intellectual exertion is one of the most common causes of cerebral softening. Eleven of the cases occurring in my experience were clearly the result of this cause. Severe and protracted emotional disturbance was apparently the cause in four cases.

Rostan, among the causes, cites insolation, the action of intense cold, blows upon the head, and excessive use of alcoholic liquors.

The influence of obliteration of the cerebral arteries, sinuses, veins, and capillaries, in producing partial cerebral anæmia, and hence as leading to the supervention of softening, has already been dwelt upon at sufficient length.

Diagnosis.—The history of hæmorrhage, thrombosis, or embolism, when these conditions have either of them given rise to softening, will aid in the diagnosis. The signs which serve to distinguish these affections from others have already been amply considered.

When there is no such previous clinical history, softening of the brain may be confounded with chronic meningitis, meningeal hæmorrhage, or tumors. From chronic meningitis it is to be distinguished in many cases by the facts that in the former the headache is generally diffused, while in softening it is fixed, that the paralysis is more limited, that there are frequent spasms of the limbs, that there are well-marked febrile exacerbations, and that there is not the progressive enfeeblement of the intellect so characteristic of the vast majority of cases of cerebral softening. At the same time it must be admitted that the diagnosis sometimes cannot be clearly made out.

In meningeal hæmorrhage coma occurs as an early symptom, gradually increasing in intensity, whereas in softening it comes on at a late period. Hæmatoma of the dura mater, however, may readily be confounded with softening. The history of the case will aid in the formation of a correct diagnosis.

In tumors the most prominent symptoms are pain and convulsions, while the intellect usually remains unaffected. The pain is exceedingly intense, while in softening it is dull. The speech in tumors is generally unaffected.

Prognosis.—Cerebral softening in general ends in death. Nevertheless, it is not altogether hopeless. If the patient be young, of good constitution, and of temperate habits; if the centre of softening be small, and not involving the more important parts of the brain, there is some encouragement to expect a favorable termination. Some of the cases cited in this chapter go to show that recovery is possible, and I have certainly seen others with the ordinary initial symptoms of cerebral softening recover with appropriate medication. Such patients, however, were all under the age of forty, and were of good constitution and habits. In softening due to embolism, and occurring after rheumatism and endocarditis, the liability to future attacks must not be overlooked. I have seen as many as six attacks of embolism occurring in the same patient, and yet no morbid condition beyond that of anæmia set up, and again cases in which a single embolus has caused softening and death.

Morbid Anatomy.—In the softening of the brain which results from the obliteration of arteries or veins by embolism or thrombosis, the first stage after that of congestion from the excessive action of the collateral circulation is what is called yellow softening. This is not, as some authors have supposed, produced by the infiltration of pus into the cerebral substance, but is caused by regressive metamorphosis of the brain-cells into fat, the granules of which are mixed with the coloring matter of the blood which gives rise to the peculiar yellow color. The white corpuscles of the blood also undergo degeneration into fat.

These altered white corpuscles were described by Gluge[1] as inflammation corpuscles, under the idea that softening was always the result of inflammation. Laborde,[2] who has studied this subject with great success, shows, however, very conclusively that the transformation is a true degeneration, a part of the fat-corpuscles being derived, as stated above, from the nervous fibres, the cylinders of which disappear, the contents being extravasated, and with the myeline being converted into fat; and another part consisting of altered white blood-corpuscles. At this time the cerebral tissue is pulpy, constituting a centre of softening or a *foyer*, the consistence of which is greater at the circumference than at the centre. The blood-vessels passing through the disorganized portion are easily separated from the perivascular tissue and are covered with oil-globules.

---

[1] "Atlas of Pathological Histology." Translated by Leidy. Philadelphia, 1853.

[2] *Op. cit.*

The second stage is designated white softening, and in it the brain-substance loses altogether its morphological characteristics, and appears as a white, cream-like matter so soft that a weak stream of water, allowed to impinge upon it, washes it away. In this semi-liquid matter, whitish flakes of denser tissue are suspended. Microscopical examination shows that all traces of nervous structure have disappeared, and that no anatomical elements remain except oil-globules and organic corpuscles somewhat resembling leucocytes.

When the morbid process involves the cortical substance of the cerebrum, the convolutions undergo a peculiar kind of transformation first pointed out by Cruveilhier, and then by Durand-Fardel[1] as occurring in the senile form of softening.

This is characterized by the formation of yellow plates, irregular in form, soft to the touch, but yet sufficiently dense to resist the action of a thin stream of water. Microscopically they are seen to consist of nucleated fibres, fat-corpuscles, fat-globules, and degenerated capillaries, with blood-crystals and granular matter. Essentially, therefore, they are formed of connective tissue.

The degenerated nerve-tissues, constituting a focus of softening, may undergo absorption. In such a case, a cicatrix, similar in general characteristics to that resulting from the curative process of hæmorrhage, remains.

In the softening resulting from inflammation, a somewhat different set of morbid appearances exists. Thrombosis and embolism produce a true death of the parts previously supplied by the occluded vessels, a necrobiosis, as it has been called by Virchow. The process is accompanied, as we have seen, by degeneration of the nervous tissue, but in the softening due to inflammation new formations result. Sometimes the two coexist, but the latter is occasionally an entirely independent action.

When such is the case, connective tissue is generated, and the nervous substance is rapidly broken down. An exudation of an albuminous fluid containing fine granules, the disintegrating nervous substance and numerous flakes of coagulated fibrine, takes place, and with blood-corpuscles causes the centre of softening to present the appearance of a reddish pultaceous mass, easily washed away by the action of a weak stream of water. With age the color of this softened tissue becomes brown or yellow. Sometimes, when the inflammation has extended to the deeper parts of the cerebrum, the contents of the cyst are penetrated by the new connective tissue. The pulpy mass undergoes partial absorption, and is replaced by a white turbid liquid, called by Cruveilhier and Dechambre "milk of lime" (lait de chaux). Durand-Fardel designates this form of softening "cellular infiltration."

---

[1] "Maladies des vieillards," Paris, 1854, p. 72.

The softening resulting from occlusion of the capillaries, a condition not recognizable during life, does not differ essentially, except in its situation, from that which follows embolism or thrombosis of the larger vessels. The centres of the process are, however, smaller, are generally numerous, and usually met with either in the cortical or white substance, or in the corpora striata. The morbid anatomy of the affected vessels has been sufficiently considered in the previous chapter.

When disease of the capillaries has been the cause of the softening, these may be ruptured, and we meet with minute extravasations of blood in the disintegrated perivascular tissue, constituting the "capillary hæmorrhage" of Cruveilhier.

Pathology.—The first definite accounts of cerebral softening were given by Lallemand [1] and Rostan,[2] both of whom published their works in the same year, 1820.

In the very beginning of his first letter, Lallemand awards to MM. Récamier, Bayle, and Cayot, the credit of describing the condition under consideration, and of giving it the designation by which it is so generally known, even out of France, of ramollissement. Lallemand then proceeds to define the term by saying that, by ramollissement of the brain, he understands a kind of liquefaction of a part of its substance, the remainder preserving its ordinary consistence. He then quotes cases from Morgagni and Abercrombie, and cites others from his own experience; and then concludes by declaring that he does not hesitate to range cerebral softening among the inflammations, in which opinion he is supported by Abercrombie.[3] Rostan [4] regarded the disease as sometimes being due to inflammation, and sometimes to degeneration of the blood-vessels. Bouillaud [5] viewed it as an anatomical feature of inflammation. Cruveilhier [6] considered what he called red softening as resulting from the capillary hæmorrhage previously mentioned, and that other forms were certainly due to inflammation.

Andral [7] recognized the fact that softening might result from inflammation or capillary hæmorrhage, but he also insisted that it might be due to special alterations of nutrition, caused by different morbid influences, such as obliteration of the arteries supplying the brain, or impoverishment of the blood.

MM. de la Berge and Monneret [8] adopted in part the views of Rostan relative to degeneration of the cerebral vessels as a cause of softening. Carswell[1] regarded softening occurring during life as being affected by these circumstances—inflammation, obliteration of arteries, and modification of nutrition.

---

[1] "Recherches anatomico-pathologiques sur l'encéphale," Paris, 1820.

[2] "Recherches sur le ramollissement du cerveau," Paris, 1820. My references to Rostan's work are to the second edition, of 1823.

[3] *Op. cit.*, p. 205.

[4] *Op. cit.*, chapter vii.

[5] "Traité de l'encéphalite," Paris, 1825.

[6] Art. "Apoplexie," in "Dictionnaire de médecine et de chirurgie pratiques."

[7] "Clinique médicale."

[8] "Compendium de médecine pratique."

Fuchs[2] appears to think that inflammation is not a necessary antecedent, but that congestion is. He also admits obstruction of the arteries at the base of the brain to be a cause.

The studies of Durand-Fardel[3] have been very thorough, and have contributed greatly to our knowledge of cerebral softening. According to him, the affection is an inflammation which does not differ essentially from other inflammations occurring in the young or old. White softening he regards as the chronic form of the disease.

Other pathologists published the results of their observations and generally to the same effect as those which have been quoted, viz., that cerebral softening was an inflammatory process, and sometimes one resulting from obliteration or disease of the arteries. A few, however, held to the view of Lallemand and Durand-Fardel, that inflammation was always the starting-point.

In 1847 Virchow published his observations relative to embolism, and the partial cerebral anæmia produced by occlusion of an artery thus became a recognized fact. In reality, it came to be regarded as the only cause capable of giving rise to softening, and many pathologists of the present day entertain such an opinion. But I think this is carrying the theory further than facts will warrant. I cannot altogether disregard the researches of Durand-Fardel,[4] Calmeil,[5] Rokitansky,[6] Wedl,[7] and others, and although I cannot agree that all cerebral softening is a consequence of inflammation, I am very sure it has this and other causes besides thrombosis and embolism. Calmeil's work is a monument of careful observations and scientific deductions, and his fifth chapter (tome ii.), entitled "*Du ramollissement cérébral local aigu, ou de l'encéphalite locale aiguë sans caillots sanguins siégeant sous la forme d'un foyer ou des plusieurs foyers circonscrits, soit à la surface, soit dans la profondeur de la masse encéphalique*," contains cases which are amply sufficient to establish the point for which he contends. He shows, too, in other chapters of his treatise, that softening results about the periphery of clots due to cerebral hæmorrhage.

The weak feature of Calmeil's otherwise very complete work is, that he altogether ignores Virchow, and those after him, who have confirmed his facts and theories.

---

[1] Art. "Softening of Organs," in "Cyclopædia of Practical Medicine," vol. iv., p. 176, American edition.

[2] "Beobachtungen und Bemerkungen über Gehirnerweichung," Leipzig, 1838.

[3] "Traité du ramollissement du cerveau," Paris, 1843.

[4] "Maladies des vieillards," Paris, 1854.

[5] "Traité des maladies inflammatoires du cerveau," Paris, 1859.

[6] "Pathological Anatomy," Sydenham Society translation, 1850.

[7] "Rudiments of Pathological Histology," Sydenham Society translation, 1855.

Soulier,[1] on the other hand, can see in softening nothing of the nature of inflammation. For him it is always a necrobiosis, produced by the cessation of the physiological action of the blood, obliteration by embolus or thrombus, by diminution of the calibre of the vessels, or occlusion resulting from atheroma or obstruction of a vein or sinus. He admits that the obliteration of an artery may cause congestion behind the point of obstruction, by which the coagulation and capillary hæmorrhage of acute softening—the capillary apoplexy of Cruveilhier—are to be explained. This red ramollissement has, however, nothing of the nature of inflammation about it.

The only points in which I differ with Soulier are, that I cannot regard softening as being solely due to occlusion of blood-vessels, and that I am very sure the congestion which follows thrombosis or embolism is not necessarily the first stage of softening. There is no more reason why partial cerebral anæmia should always result in softening, than that ligation of the femoral artery should always lead to gangrene of the parts below.

Obstruction of veins and sinuses in the brain may be followed by softening. The clot is usually the result of injuries or disease of the cranial bones or cerebral membranes, especially the dura mater. It may also be caused by certain cachectic conditions in which the blood is deteriorated in quality, such as typhus and typhoid fevers and cholera.

Four cases, in which this latter affection was followed by thrombosis of the superior longitudinal sinuses, with consecutive softening, have come under my observation. In two of them there were also thrombi in both femoral veins. The upper surfaces of both hemispheres were the seats of the softening, which involved the gray matter only.

Thrombosis of the veins or sinuses may also in general terms be produced by whatever cause is capable of retarding the current of blood. Mr. Toynbee,[2] in his chapter on diseases of the mastoid cells, has brought forward several cases in which the lateral sinus was occluded by coagula, and in which there was cerebral softening.

Cerebral softening may also result from the formation of adventitious growths, or from the presence of foreign bodies in the brain. In such cases the process begins with inflammation, and is similar to the action which sometimes goes on around an extravasation of blood.

Acute cerebritis or meningitis may likewise result in softening. This fact is admitted by Drs. Russell Reynolds and Bastian, in their admirable essays on cerebritis and softening of the brain, though with evident reluctance.

We see, therefore, that cerebral softening may be caused either by anæmia or inflammation, and that it is of two kinds, inflammatory and non-inflammatory. The seat of the softening may be in any part of the brain, although some regions are more liable than others. When due to thrombosis, there appears to be no predilection for any particular location, but, as embolism is generally found on the left side in the middle cerebral artery, the parts of the brain supplied by this vessel are more liable than the corresponding parts of the right side.

---

[1] *Journal de médecine de Lyon*, Février, 1867.

[2] "The Diseases of the Ear, their Nature, Diagnosis, and Treatment," London, 1860.

Durand-Fardel, however, did not arrive at this conclusion. Of one hundred and sixty-nine cases of softening, he found the left hemisphere the seat in sixty-nine, the right in seventy-one, both in twenty-six, and the middle line in three.

The gray matter is generally supposed to be more frequently the seat of softening than the white. It is true that, of thirty-three cases of acute softening observed by Durand-Fardel,[1] the convolutions were involved in thirty-one, but in nine only were they the sole part affected.

In fifty-three cases which the same author collected from the writings of Rostan, Lallemand, and others, the centres of softening were found to be as stated in the following table. Occasionally more than one region was involved.

Convolutions and white substance	22
Convolutions alone	6
White substance alone	5
Corpus striatum and optic thalamus	6
Corpus striatum alone	11
Optic thalamus alone	4
Pons Varolii	3
Crux cerebri	1
Corpus callosum	1
Walls of the ventricles (septum)	1
Fornix	1
Cerebellum	1

Rostan, on the other hand, found the corpora striata and the optic thalami to be the parts most frequently affected, and after these the central part of the hemispheres. He met with but few cases involving the median line.

As regards the frequency with which the convolutions with the white substance were involved, as compared with the motor tract, he found that, of one hundred and seventy-seven cases of acute and chronic softening, the convolutions and white substance were affected in one hundred and nineteen, and the corpora striata and optic thalami in fifty-eight.

The middle lobe is more liable than any other, as is seen in the following statement of Durand-Fardel, based upon an analysis of ninety-five cases:

---

[1] "Traité du ramollissement du cerveau," Paris, 1843.

Posterior lobe	18
Middle	51
Anterior	13
Posterior and middle	7
Posterior and anterior	2
Middle and anterior	2
Whole convexity of hemisphere	1
Middle line	1

In more than one-half of the cases, therefore, the middle lobe was the seat of the disease.

A question connected with the pathology of cerebral softening, as with hæmorrhage, is, "Can we determine, from a consideration of the symptoms, what part of the brain is the seat of the lesion?" The answer must be the same. We can do so with some approach to accuracy, but, till we are better acquainted with the physiology of the different ganglia composing the brain, we cannot expect to do so with absolute certainty. Indeed, owing to the greater extent of tissue involved, compared to that affected in hæmorrhage, we have a more complicated set of phenomena to deal with. I have nothing further to add to the remarks made on a similar point, under the head of cerebral hæmorrhage.

Treatment.—The treatment proper for cerebral softening should depend very much upon the cause from which it has arisen, and must more or less be directed against the symptoms which are manifested. Thus, if there is reason to suspect the existence of thrombosis or embolism, and a consequent anæmic condition of a portion of the brain, the judicious use of stimulants and tonics is advisable, while the body should be kept warm by additional clothing, or the application of artificial heat—at the same time the recumbent posture should be assumed, and the head supported on a low pillow. Mental exertion should, of course, be absolutely interdicted. If there be much headache, it is probably due to too great an activity of the collateral circulation, and in such a case some one of the bromides may be given in large doses, repeated as often as may be necessary. I have frequently seen great relief follow their administration.

Delirium is often due to a like cause and may be similarly treated. Dr. Reynolds[1] speaks highly of the Indian hemp in doses of a quarter to half a grain of the extract; but I have found the bromide of potassium, in doses of thirty grains every three or four hours, more efficacious. It is also the most beneficial remedy in the convulsions which frequently precede a fatal termination.

In that form of softening which is obscure in its origin and gradual in its progress, there is a little more hope of a favorable result, though even here it must be confessed that treatment is not often effectual.

---

[1] Article, "Softening of the Brain," in "System of Medicine," vol. ii.

Still, as I have said, when speaking of the prognosis, there are undoubtedly cases in which recovery has taken place, and I am very sure that I have several times succeeded in curing individuals who, so far as I have been able to judge, were affected with cerebral softening. As these cases are interesting in themselves, and as the histories will show the means of treatment employed, I do not hesitate to transcribe the following typical ones from my case-book:

I.—Mr. R., a gentleman, twenty-four years of age, awoke one morning about the middle of March, 1870, with a sensation of numbness extending through the whole of the left arm and leg, and with a feeling of vertigo which was insupportable when he arose from the bed. He sat down in a chair, and while in this position was conscious of a buzzing sound in the right ear. In the course of half an hour the vertigo passed off, but the numbness and sound in the ear remained, and he occasionally saw double. In a few days afterward he noticed a slight difficulty of articulation, owing to apparent thickness of the tongue, and about the same time observed that in the morning the pillow was wet with the saliva which had run from his mouth during sleep. His uncle, a wealthy gentleman of this city, sent him off traveling, but he returned in a few weeks with loss of power in the left arm and leg, which had begun to be manifested to a slight extent before his departure. He came under my charge May 15, 1870.

At this time the paralysis, of both motion and sensation, was well marked on the left side, as shown by the æsthesiometer and dynamometer. The line made by the dynamograph with the right hand was perfectly straight, while that made by the left was at an angle of forty-five degrees with the other. In his conversation he clipped his words, and sometimes left out the smaller ones. His memory he stated was materially impaired. There was almost constant headache over the whole frontal region, and attacks of vertigo were frequent. There was no marked paralysis of the face, though the muscles of both sides were paretic, and he often had double vision. The right pupil was largely dilated and was insensible to light.

Ophthalmoscopic examination showed the left eye to be perfectly normal, but the retinal vessels of the right were smaller and straight, and the choroid was paler than natural.

Upon inquiry I ascertained that he had given extraordinary attention to his business for a period of several months before the attack of numbness, frequently being up making calculations till three o'clock in the morning, and thus depriving himself of the necessary amount of sleep.

My opinion was, that he was suffering from incipient softening of the brain due to disease of the capillaries, which, in its turn, resulted from cerebral congestion and exhaustion. I was further of the opinion that the lesion involved the right hemisphere and motor tract.

I prescribed the phosphide of zinc in the dose of the tenth of a grain, with half a grain of extract of nux-vomica in pill three times a day, with the constant galvanic current three times a week, the latter to be derived from fifteen of Smee's cells, and to be passed from forehead to occiput for three or four minutes at a time. At the end of ten days he had lost his diplopia, the pupil of the right eye had regained its natural diameter and irritability, and the vertigo and headache had notably diminished. The treatment was continued, and at the end of a month he had recovered the sensibility and power on the paralyzed side to such an extent, and had improved so much in other respects, that I advised him to take a short journey. He was absent two weeks, during which period he continued to take the pills as before, and on his return was, to all appearance, well. He has since remained in excellent health.

II.—Mr. R. W., a merchant of this city, consulted me in April, 1868, under the following circumstances:

After a long period of great domestic anxiety, during which he had been engaged in some heavy commercial transactions, and had suffered from wakefulness, he experienced one afternoon, while riding in the park in his carriage, a slight quivering motion at the apex of the tongue. It continued until he reached home; and then, upon looking in a mirror, he could see the fibrillary movement very distinctly. He was not alarmed, and went to bed at his usual hour. In the morning he noticed a little thickness of speech, but the movement had ceased. That afternoon he had a violent headache, attended with vertigo and nausea. Becoming alarmed, he sent for his family physician, who ascribed the symptoms to indigestion, and administered a mild cathartic. The following day, on attempting to rise from the bed to go to the water-closet, he was attacked with such a severe vertigo that he was obliged to lie down again; and, though he did not for a moment lose consciousness, his fæces escaped from him involuntarily. From this time he gradually lost strength in both arms and legs, and his speech became very defective. His memory suffered to such an extent that he forgot the names of his children. There was very little headache, the vertigo had ceased, there was no disturbance of vision, and no loss of power over the sphincters. About six weeks after the occurrence of the first symptom noticed, he came under my care.

At this time there was anæsthesia of both sides of the body, both legs and both arms had lost power; he clipped his words, and frequently substituted others of similar sound or meaning for those he ought to have used. His memory was much weakened, and there was a strong tendency to stupor. There were no troubles of the special senses—ophthalmoscopic examination revealed nothing abnormal—there was no facial paralysis. I diagnosticated softening of the brain from general cerebral anæmia consequent upon congestion and cerebral exhaustion, and I prescribed a liberal allowance of wine, a full and nutritious diet, carriage exercise, and amusements of various kinds. This was the very reverse of the treatment to which he had been subjected. In addition, I recommended the constant galvanic current, to be applied as in the previous case, and gave the following prescription: ℞. Olei phosphorat. ℥ss; mucil. acaciæ, ℥j; ol. bergamii, gtt. xv. M. ft. emulsio. Dose, gtt. xv. ter die.

The treatment was carried out with the result of obtaining a gradual and permanent improvement, so that at the end of about six months the patient was well. He then went to Europe, where he now is, with as good health as he has ever enjoyed.

Other cases, similar in their general features, have been under my care with a like result in each, and several others have been very decidedly improved and relieved of the more prominent symptoms of the disease, without, however, regaining full health. The means of treatment thus far consist in the use of tonics, stimulants, and especially phosphorus and strychnine, the avoidance of all severe mental exertion, and all excessive emotion, open-air exercise, and the use of the constant galvanic current.

The beneficial effects of maintaining the physical strength were several years since pointed out by Mr. F. Skey[1] in a clinical lecture delivered at St. Bartholomew's Hospital, but it must be confessed that the opposite plan of treatment has been very generally followed.

Softening from the effects of thrombosis or embolism is, as I have said, not much under the control of the physician. Patients recover from it, however, when they are of good constitution, and when the focus of softening has not been extensive. The mind and body may, and in such cases generally do, remain feeble, and we are therefore consulted for the relief of the condition. In such cases tonics, and among them phosphorus, strychnine, and wine, occupy a prominent place; the constant galvanic current to the head, and the induced to the paralyzed muscles, will rarely fail to be of service.

III.—Thus a gentleman, who had been a distinguished officer of the army, suffered from loss of memory, defective articulation, ptosis, double vision, and right hemiplegia, probably the result of embolism. Several years before he came under my charge, he had been treated by Dr. J. T. Metcalfe, for heart-disease, the result of acute rheumatism. I gave the phosphide of zinc and extract of nux-vomica according to the formula previously mentioned, advised a liberal use of wine and beefsteaks, applied the primary current to the brain, and the induced current to his paralyzed arm and leg, and in a few weeks had the satisfaction of seeing such a degree of improvement as almost to constitute a cure. The ocular troubles had disappeared, his memory had improved, he talked as well as ever, and the numbness and loss of strength were no longer remarked unless he over-exerted himself, which, owing to his general feeling of *bien aise*, he was very apt to do. He remained in this condition for over a year, when he had several other attacks of embolism, each of which left him more weak, mentally and physically, than before, and of which he eventually died.

---

[1] "On the Value of Tonic Treatment in some Diseases of the Brain, more especially Cases of Ramollissement," *Dublin Hospital Gazette*, November, 1858.

There were some interesting features connected with this case, which will be referred to at greater length under the head of aphasia.

IV.—In another case, in which there was reason to think a *foyer* of softening had been absorbed, a marked relief from the sequelæ was obtained. The patient, a literary gentleman of distinction, had, several years previously to my seeing him, suffered from an attack of acute rheumatism with endocarditis. About a month after his recovery, as he was sitting in his library before the fire, he felt a sensation as if one side of his face had suddenly become much heavier than the other. Almost immediately afterward he lost consciousness, and fell to the floor. He could not have been in this condition longer than five minutes when he came to himself, to find that he was paralyzed in the right arm and leg. Attempting to call for assistance, he found he could not articulate. His wife soon afterward entered the room, and medical aid was obtained. He was bled to the extent of sixteen ounces, and purged with croton-oil.

The following day he was much better; could move his arm and leg, and articulate with some degree of distinctness, but toward evening headache ensued, he became delirious, and the paralysis increased. Of the condition immediately following, he could give no very clear account. He only knew that he was confined to his bed for several weeks, was delirious part of the time, and that, after the acute attack passed off, he was left with an enfeebled mind, imperfect articulation, and paralysis of the arm and leg on the right side. He went to Europe, traveled extensively, and returned at the end of a year very much improved, but still with some degree of mental weakness, defective speech, and paralysis, remaining.

When he came under my observation, the following were the principal symptoms observed: The strength of the right arm, as measured with the dynamometer, was not one-third that of the left; the extensors of the leg and foot were almost entirely paralyzed, so that in walking he abducted the leg so as to cause the foot to clear the ground; electro-muscular contractility was much weakened, though the induced current caused feeble contractions. His speech was affected mainly as regarded the memory of words. He spoke with a good deal of volubility, but constantly used the wrong expressions. Thus, when he wished to tell me that he had visited Europe for the benefit of his health, he said: "I went to Elope for the bequest of my hedge," and then went on—continually making other mistakes—to tell me a long story which I could scarcely understand. His emotions were easily disturbed: he cried because he had to wait a few minutes in my reception-room before seeing me.

Ophthalmoscopic examination showed pale choroids and straight and attenuated retinal vessels. Auscultation revealed the existence of both mitral and aortic regurgitation.

Taking into consideration the history of the case and the present condition of the patient, I diagnosticated embolism of the left middle cerebral artery, subsequent softening and eventual absorption of the diseased part of the brain. My idea was that the brain, as a whole, was anæmic, and that, with improved nutrition of it and the paralyzed limbs, amelioration of the symptoms was possible.

I therefore prescribed the phosphide of zinc and nux-vomica pills as before mentioned, directed the use of wine to the extent of half a bottle of champagne daily, and advised that animal food should form the principal portion of each meal. Since his illness he had, by direction of his physician, left off the use of coffee. I directed it to be resumed, and to be taken strong. The primary galvanic current was passed through the head in the manner previously indicated in this chapter, and the induced current was applied for half an hour three times a week to the arm and leg, each paralyzed muscle receiving a full share of attention.

It was not long before signs of amendment were noticed. His strength became greater in the arm, and he was able to extend the leg and to raise the foot after half a dozen electrical applications. His speech next gave evidence of improvement, and his mind became stronger. The treatment was continued for about four months, with only an intermission of a week. At the end of that time his gait was almost natural, though he still swung the foot a very little, his arm was nearly as strong as the other, his mind was not perceptibly weaker than that of other persons of his age (fifty-five), and his speech was excellent except when he was excited and very anxious to express himself correctly and fluently.

There is one point in regard to which a few words are perhaps necessary, and that is to enter a protest against the use of counter-irritation of any kind, and to discountenance, as far as I can, the employment of the actual cautery. I have never seen the least advantage follow the application of croton-oil to the shaven scalp, nor can I conceive how such a measure can be recommended on rational grounds. I have several times witnessed its action, and have invariably seen it aggravate the symptoms. In the case of a gentleman from St. Louis, affected with cerebral softening, the effect was to make his speech still more imperfect and his mind weaker. A lady, who was affected with all the more prominent symptoms of softening of the brain, had all the phenomena increased in violence after the application of the actual cautery to the nape of the neck. I could easily adduce other examples to the same effect, were it necessary.

CHAPTER VII.

APHASIA.

The subject of aphasia is of such interest, and so much attention has recently been given to it by physiologists and pathologists, that, although it is only a symptom common to several morbid conditions, a treatise on diseases of the nervous system would scarcely be regarded as complete without its being fully considered.

By aphasia is understood a condition produced by an affection of the brain by which the idea of language, or of its expression, is impaired. The word is derived from the Greek—*a*, privative, and *φασις*, speech—and, as stated by Trousseau, was proposed by M. Chrysaphis, a distinguished Greek scholar, as a substitute for *alalia*, used by Lordat, and *aphemia*, employed by Broca, to designate the same condition.

In the definition which I have given of aphasia, the term is limited to impairment of the idea of language or of its expression. It does not, therefore, include those cases in which the individuals are able to speak, but will not; such as are met with among the insane. The idea of language is as perfect as ever, and is doubtless entertained, but the person does not speak because he does not will to do so, and this failure may arise either from a lack of the necessary power, or from a stubborn determination not to speak. A lady was a short time since under my charge who had been treated by a homœopathic physician as a case of aphasia. A very slight examination was sufficient to convince me that the case was one of hysteria. She had not spoken for several months, but upon one occasion she came to my office with her maid, whom she required to repeat the alphabet, and when the right letter was reached she signified the fact by raising her hand. She thus spelled out the words she wished to use. Subsequently she procured a card with all the letters on it, such as are used for children learning their alphabet, and she composed her words from this. Of course all these facts showed that her idea of language was intact, but she still might have lost the power of coördinating the muscles concerned in articulation so as to express herself in spoken words. Although I was sure this was not the case, I failed to make her speak, until one morning she became very much interested in something I was saying, and, finding her alphabet too slow a means of expression, dropped it and began to speak with great fluency. After talking with energy for a quarter of an hour, she suddenly recollected herself and took up her card of letters again, but the charm was broken, and by degrees she resumed her speech. At one time this lady was under the care of my friend Prof. Flint, for some chest or throat difficulty, and on one occasion spoke very well.

Neither does aphasia embrace cases of inability to speak from paralysis of the tongue or other muscles of articulation. Defective speech from this cause is frequently met with in hemiplegia, in glosso-labio-laryngeal paralysis, and some other affections. In such instances the idea of language remains, but the patient does not speak because he is unable to put the organs of articulation in motion. A few days ago a gentleman, a prominent merchant of the city, was sent to me as a case of aphasia. As he entered my consulting-room, I saw that he was hemiplegic on the left side, and, on telling him to put out his tongue, found that he could not get it beyond the teeth, or touch the roof of his mouth with it. The history of the case was that of ordinary cerebral hæmorrhage, and he regained the power of speaking after several applications of the primary and induced galvanic currents had been made to the tongue and muscles of the face.

The distinction between aphonia and aphasia must also be made. In the one the idea of speech is undisturbed, and articulation is not interfered with except as regards phonation. Aphonic patients can whisper, but are unable to speak in full voice, owing to some laryngeal affection impairing the tone of the vocal chords.

The fact that the faculty of speech may be deranged independently either of the will, paralysis, or loss of voice, appears to have been noticed at a very early period in the progress of science. Thus Isaiah[1] says, "For with stammering lips and another tongue will he speak to this people;" and again,[2] "Thou shalt not see a fierce people, a people of a deeper speech than thou canst perceive; of a stammering tongue that thou canst not understand."

Thucydides mentions that many, who suffered from the plague which raged at Athens, found on recovering that they had not only forgotten the names of their friends and relations, but also their own names.

Pliny,[3] in the chapter entitled *Memoriæ Exempla*, says, in speaking of this faculty: "For nothing is so weak in man; disease, falls, injuries, even a fright, may impair it partially, or destroy it altogether. A blow from a stone has abolished the memory of the alphabet. A fall from a high roof has caused a man to cease to recognize his mother and neighbors, another even forgot his slaves, and Messala Corvinus, the orator, could not recall his own name."[4]

---

[1] Chapter xxviii., 11. [2] Chapter xxxiii., 19. [3] Lib. vii., cap. xxiv.

[4] Trousseau has translated this passage somewhat differently. I quote from an illuminated copy printed at Tarvisium (Treviso), in October, 1479.

Suetonius[1] relates that Claudius so far lost his memory that he forgot the names of persons to whom he desired to speak, and could not even recollect the words he wished to use.

Passing over several authors of later times who have recognized the existence of the difficulty in question, we come to Crichton,[2] who remarks as follows: "There is a very singular defect in memory, of which I have myself seen two remarkable instances. It ought rather to be considered as a defect of that principle by which ideas and their proper expressions are associated, than of memory, for it consists in this, that the person, although he has a distinct notion of what he means to say, cannot produce the words which ought to characterize his thoughts. The first case of this kind which occurred to me in practice was that of an attorney much respected for his integrity and talents, but who had many sad failings to which our physical nature too often subjects us. Although nearly in his seventieth year, and married to an amiable lady much younger than himself, he kept a mistress, whom he was in the habit of visiting every evening. The arms of Venus are not wielded with impunity at the age of seventy. He was suddenly seized with great prostration of strength, giddiness, forgetfulness, insensibility to all concerns of life, and every symptom of approaching fatuity. His forgetfulness was of the kind alluded to. When he wished to ask for any thing, he constantly made use of some inappropriate term. Instead of asking for a piece of bread, he would probably ask for his boots; but, if these were brought, he knew they did not correspond with the idea he had of the thing he wished to have, and was therefore angry. Yet he would still demand some of his boots and shoes, meaning bread. If he wanted a tumbler to drink out of, it was a thousand to one he did not call for a certain chamber-utensil, and, if it was the said utensil he wanted, he would call it a tumbler or a dish. He evidently was conscious that he pronounced wrong words, for, when the proper expressions were spoken by another person, and he was asked if it were not such a thing he wanted, he always seemed aware of his mistake, and corrected himself by adopting the appropriate expression. This gentleman was cured of the complaint by large doses of valerian and other proper medicines."

Dr. Crichton subsequently met with another case similar to the foregoing, and he quotes the following from Prof. Gruner, of Jena, in vol. vii. of the *Psychological Magazine*. The patient, a learned gentleman, after his recovery from an acute fever, suffered a loss of memory for words. Among the first things he desired to have was coffee (*kaffee*), but, instead of pronouncing the letter *f*, he substituted in its place a *z*, and therefore asked for a cat (*kazze*). In every word which had an *f* he committed a similar mistake, substituting a *z* for it.

---

[1] "C. Suetonii Tranquilli," xii. Cæsares.

[2] "An Inquiry into the Nature and Origin of Mental Derangement, comprehending a Concise System of the Physiology and Pathology of the Human Mind, and a History of the Passions and their Effects," London, 1798, vol. i., p. 371.

He also cites, from Van Goens, the case of Madame Hennert, wife of the professor of mathematics at Utrecht, who suffered a similar defect of memory. When she wished to ask for a chair she asked for a table, and when she wanted a book she demanded a glass. But, what was singular in her case was, that when the proper expression of her thought was mentioned to her, she could not pronounce it.

She was angry if people brought her the thing she had named instead of the thing she desired. Sometimes she herself discovered that she had given a wrong name to her thoughts. This complaint continued several months, after which she gradually recovered the right use of her recollection. It was only in this particular point that her memory seemed to be defective, for M. Van Goens assures us that she conducted her household affairs with as much regularity as she ever had done, and that she used to show her husband the situation of the heavens on a map with as much accuracy as when she was in perfect health.

The following case, in Gesner's *Endeckungen der Neuesten Zeit in der Arzneigelehrheit*, is likewise quoted by Crichton:

"A man, aged seventy, was seized, about the beginning of January, with a kind of cramp in the muscles of the mouth, accompanied with a sense of tickling all over the surface of the body, as if ants were creeping over it. On the 20th of the same month, after having experienced an attack of giddiness and confusion of ideas, a remarkable alteration of his speech was observed to have taken place. He articulated easily and fluently, but made use of strange words, which nobody understood. The number of these does not at present seem to be great, but they are frequently repeated. Some of them he seems to forget entirely, and then new ones are formed. When he speaks quick he sometimes pronounces numbers, and now and then he employs common words in their proper sense. He is conscious that he speaks nonsense. What he writes is equally faulty with what he speaks. He cannot write his name. The words he writes are those he speaks, and they are always written conformably to his manner of pronouncing them. He cannot read, and yet many external objects seem to awaken in him the idea of their presence."

Dr. Rush,[1] in the work the title of which is cited below, in chapter xii., which treats *of Derangement in the Memory*, refers so specifically to affections of the speech that I quote his language with some degree of fullness, and I do so with the less hesitation as his observations appear to have escaped notice, both in this country and in Europe. He says:

"1. There is an oblivion of names and vocables of all kinds.

"2. There is an oblivion of names and vocables, and a substitution of a word no ways related to them. Thus, I knew a gentleman afflicted with this disease, who, in calling for a knife, asked for a bushel of wheat.

---

[1] "Medical Inquiries and Observations upon Diseases of the Mind." Fourth edition. Philadelphia, 1830, p. 274. The first edition was published in 1812.

"3. There is an oblivion of the names of substances in a vernacular language, and a facility of calling them by their proper names in a dead or foreign language. Of this, Wepfer relates three instances. They were all Germans, and yet they called the objects around them only by Latin names. Dr. Johnson, when dying, forgot the words of the Lord's prayer in English, but attempted to repeat them in Latin. Delirious persons, from this disease of the memory, often address their physicians in Latin or in a foreign tongue.

"4. There is an oblivion of all foreign and acquired languages, and a recollection only of vernacular language. Dr. Scandella, an ingenious Italian, who visited this country a few years ago, was master of the Italian, French, and English languages. In the beginning of the yellow fever which terminated his life in the city of New York in the autumn of 1798, he spoke English only; in the middle of his disease he spoke French only; but on the day of his death he spcke only in the language of his native country.

"5. There is an oblivion of the *sound* of words, but not of the letters which compose them. I have heard of a clergyman in Newburyport, who, in conversing with his neighbors, made it a practice to spell every word that he employed to convey his ideas to them.

"6. There is an oblivion of the mode of spelling the most familiar words. I once met with it as a premonitory symptom of palsy. It occurs in old people, and extends to an inability, in some instances, to remember any more of their names than their initial letters. I once saw a will subscribed in this way by a man in the eightieth year of his age, who during his life always wrote a neat and legible hand.

. . . .

"9. There is an oblivion of names and ideas, but not of numbers. We had a citizen of Philadelphia many years ago, who, in consequence of a slight paralytic disease, forgot the names of all his friends, but could designate them correctly by mentioning their ages, with which he had previously made himself acquainted."

Dr. Rush remarks of these cases, that "there appears to be something like a palsy of the mind, *quoad* these specific objects."

Thus far there had been no attempt to define with precision the seat of the faculty of language, or even to establish its existence; but, in the early part of the nineteenth century, Dr. Gall, a German physician, announced that such a faculty did exist, and that it was seated in those convolutions of the brain which rest upon the posterior part of the supra-orbital plate, and that a large development of the organ was indicated by prominence and depression of the eyes. He was first led to believe in the existence of such an organ by observing that some of the scholars with whom, as a young man, he had to compete, excelled him n the ability to learn by heart, and he noticed that those thus endowed ith great memory for words possessed prominent eyes. From these ircumstances, he was gradually carried on to the foundation of his hrenological system.

In reality, however, Gall considered that there were two organs of nguage in each hemisphere—the one originating the idea of words, e other the talent for philology, and for acquiring the spirit of lan- uages. The former organ he describes as lying on the posterior half the supra-orbital plate, as before mentioned. It gives a talent for arning and recollecting words, and persons possessing it large, recite ng passages by heart after reading them once or twice. The other is laced on the middle of the supra-orbital plate, and when it is large the yeball is not only rendered prominent but is depressed, causing the ower eyelid to assume the appearance of a bag or fold. Persons having his organ large have not only an excellent memory for words, but a articular talent for the study of languages, for criticism, and in gen- ral terms for all that has reference to literature.

Dr. Spurzheim, however, admits but one organ, lying transversely n the posterior portion of the supra-orbital plate, and this view is ac- epted by Combe and other distinguished phrenological authorities.[1]

In support of his theory that there is such an organ, Gall cites the ase of a notary reported by Pinel.[2] The latter, in speaking of apo- plexy, says this affection may be limited in its action to the words which re used to express ideas. In the case mentioned, the patient forgot, fter an attack of apoplexy, his own name, that of his wife, those of his children and friends, although there was not the least paralysis of his tongue. He no longer knew how to read or write, and yet his memory as regarded other things was unimpaired.

Dr. Gall[3] refers also to the case of a soldier, sent to him by Baron Larrey, who was affected in a manner similar to that of the notary. It was not his tongue which was involved, for he was able to move it bout in all directions, and to pronounce words, but he had lost the emory for words, although he recollected other things as well as ever.

I shall presently have occasion to refer to a still more interesting ase, reported by Larrey, and one which appears to have escaped the otice of all writers on the subject of aphasia.

Spurzheim mentions the case of one Lereard, of Marseilles, who, aving received a blow from a foil on the eyebrow (which one is not tated), lost the memory of proper names entirely. He sometimes even orgot the names of his intimate friends, and even of his father.

---

[1] For a full account of the subject, the reader is referred to a "System of Phrenol- gy," by George Combe, Boston, 1834, or to "Phrenology," etc., by J. S. Spurzheim, oston, 1833.

[2] "Traité médico-philosophique, sur l'aliénation mentale." Second edition. Paris, 809, p. 90.

[3] "Physiologie du cerveau," vol. iv., p. 84.

Gall, therefore, located the organ of language in a limited part of the anterior lobe of each hemisphere; but he adduced very little evidence to support his opinion, and hence his views did not meet with any thing like general acceptance. A number of cases, however, reported by Lallemand, Rostan, and others, support it, while several adduced by the same authors are opposed to it.

In 1825 Bouillaud,[1] who had collected a great number of cases of affections of the brain, was surprised to find how frequently the loss of speech coexisted with disease or injury of the anterior lobes. He also confirmed, what others before him had noticed, that the loss of the power of expressing ideas in articulate language was often the only evidence of a brain-affection.

He made one very important step in advance, and his views on this particular point are adopted—and often without credit—by the majority of the present writers on aphasia; he divided the faculty of speech into two distinct categories of phenomena:

1. The faculty of creating words as representatives of our ideas, and of recollecting them—internal speech.

2. The power of coördinating the movements necessary for the articulation of these words—external speech.

This classification forms the basis of the division of aphasia into the two varieties, the amnesic and the ataxic.

The cases which Bouillaud adduced in support of his theory were many of them in patients who exhibited no other symptoms than the loss of the power of articulate language. They preserved their intelligence, comprehended perfectly questions put to them, and knew the value of words; but, although there was no paralysis of either the tongue or the lips, they were unable to utter a word. At the post-mortem examination, the lesion was always found in the anterior lobes. Sixty-four cases formed the basis of his conclusions. A part was direct, and went to show that lesion of the anterior lobes was accompanied by derangement in the faculty of speech; the other part was indirect, and established the fact that, when the anterior lobes were not affected, the lesion being in some other region of the brain, the faculty of speech remained intact.

Cruveilhier opposed Bouillaud's views, and, in a paper read at the Athénée de Médecine in the same year, brought forward seven cases of persons, some of whom had lost the faculty of speech, but who, on post-mortem examination, were found to have no disease of the anterio lobes; and others who had spoken, but in whom there were more or les profound changes in these parts.

Subsequently Andral[2] reported the results of the analysis of thirt seven cases of lesion of one or both anterior lobes. Of these, speech was abolished twenty-one times, and preserved sixteen times. Lallemand[1] also opposed Bouillaud with several cases; but the latter rejoined[2] with a fresh array of thirteen cases in support of his doctrine, and with many arguments against the validity of those brought against him. Longet[3] declares that Bouillaud appears to have refuted many of the objections of his adversaries, and to have demonstrated that some of their cases were badly interpreted. At the same time, while admitting that it is possible that different parts of the brain preside over different voluntary movements, he affirms that there is nothing positively established as regards the localization of the active principles of these movements.

---

[1] "Traité de l'encéphalite," Paris, 1825; and also, "Recherches cliniques, propre démontrer que la perte de la parole correspond à la lésion des lobules antérieurs du veau," *Archives de méd.*, 1825.

[2] "Clinique médicale," tome ii., p. 135.

Subsequently, in other memoirs, Bouillaud brought forward additional cases in support of his theory, making a total of one hundred and three, and offered a prize of five hundred francs to any one who would adduce an instance of profound lesion of the anterior lobes without troubles of speech. Many years subsequently Velpeau announced that he should claim this prize, for that, in March, 1843, he had related the case, and presented the brain, of a wig-maker who had come under his care for prostatic disease. This man was in full possession of his reasoning faculties, and, moreover, was noted for his unconquerable loquacity. He died a few days subsequently, and on post-mortem examination a scirrhous tumor was found to have entirely taken the place of the two anterior lobes of the brain. Very little faith seems to have been put by physiologists or pathologists in the history of this case. If it proves any thing, it is that the anterior lobes are useless appendages to the rest of the cerebral system.

But Bouillaud was not content with the deductions to be drawn from pathology. In a series of experiments, he endeavored to establish the truth of his idea, and thus bring the science of physiology to his support. These experiments were detailed in a paper[4] read before the Academy of Sciences, in September, 1827, which was subsequently (1830) published in the tenth volume of Magendie's *Journal de Physiologie*, from which I quote.

The experiments relative to the anterior lobes were made on dogs. Only one was entirely successful—the animals in the others dying too soon after to admit of satisfactory deductions being made. But the twentieth experiment was more satisfactory.

On the 28th of June, 1826, he passed a gimlet through the anterior part of the brain of an active, docile, and intelligent dog. Immediately afterward the animal was convulsed, and could not rise from the ground. Sight and hearing remained. Symptoms of compression soon came on; the result, probably, of the hæmorrhage. Eventually, the animal recovered, but it was found to have lost much of its intelligence and agility. The faculty of memory seemed to have been entirely abolished, and there was a decided expression of imbecility in its countenance. It could no longer ascend or descend a staircase; the fore-legs were lifted very high in walking, and its movements were all badly coördinated. When struck or made to walk, it uttered sharp cries, but it had lost entirely the ability to bark. As Bouillaud remarks, "it no longer barked, either to show its affection, or to drive away strangers who came to the house." Once only, on the 18th of July, it tried to bark at a passer-by, but failed in the attempt.

---

[1] *Op. cit.*, lettres 6, 7, 8.

[2] "Exposition de nouveaux faits à l'appui de l'opinion qui localise dans les lobes antérieurs du cerveau le principe législateur de la parole." "Bulletin de l'Académie de Médecine," 1839, tome iv., p. 282.

[3] "Traité de la physiologie," tome ii., p. 438.

[4] "Recherches expérimentales sur les fonctions du cerveau (lobes cerebraux) en général et sur celles de sa portion antérieure en particulier."

This is the only experiment I have been able to find which has any bearing upon the question of the localization of the faculty of language. And I do not quote it as proving much on the subject. The difficulties in the way of experimentation are almost insuperable, to say nothing of the fact that it is doubtful if any of the sounds made by animals can be compared with human speech.

But unintentional experiments have been performed upon the human subject, which tend to show that, though the faculty of language may be located in one or both anterior lobes, either may be seriously injured without the faculty of language suffering to any appreciable extent. Two of them have happened in this country, and, although referred to in connection with aphasia by Seguin and Harris, I take satisfaction in bringing them forward on account of their great importance to the question under consideration.

The first is related by Dr. Harlow,[1] of Vermont:

The subject was a strong, healthy man, twenty-five years of age, and was engaged in ramming down a charge of powder in a rock to be blasted, when an explosion took place, and the tamping-iron was driven clear through his head.

In a few minutes he recovered his consciousness, was put into a cart and carried three-quarters of a mile to his residence, where he got out and walked into the house. Two hours afterward he was seen by Dr. Harlow. He was then quite conscious and collected in his mind, but exhausted by extensive hæmorrhage from the hole in the top of his head. Blood, pus, and particles of brain, continued to be discharged for several days, but by January 1, 1849, the wound was quite closed and his recovery complete. There was no pain in the head, but a queer feeling, which he could not describe. As regarded his mind, he was fitful and vacillating, though obstinate, as he had always been. He ecame very profane, never having been so before the accident. He ived till May 21, 1861, twelve and a half years subsequent to the accient, when he died, after having had several convulsions. His cranium as obtained, and, with the bar, is now preserved in the Warren Anaomical Museum at Boston. Dr. J. B. S. Jackson[1] thus describes the kull:

---

[1] *Boston Medical and Surgical Journal*, December, 1849, vol. xxxix., p. 389. Also, "Descriptive Catalogue of the Warren Anatomical Museum," Boston, 1870, p. 145.

"The whole of the small wing of the sphenoid bone upon the left ide is gone, with a large portion of the large wing, and a large porion of the orbital process of the frontal bone, leaving an opening in he base of the skull two inches in length, one inch in width posteriorly, and tapering gradually and irregularly to a point anteriorly. This opening extends from the sphenoidal fissure to the situation of the frontal sinus, and its centre is an inch from the median line. The optic foramen and the foramen rotundum are intact. Below the base of the skull the whole posterior portion of the upper maxillary bone is gone. The malar bone is uninjured; but it has been very perceptibly forced outward, and the external surface inclines somewhat outward from above downward. The lower jaw is also uninjured. The opening in the base, above described, is continuous with a line of old and united fracture that extends through the supra-orbitary ridge in the situation of the foramen, inclines toward and then from the median line, and terminates in an extensive fracture that was caused by the bar as it came out through the top of the head. This fracture is situated in the left half of the frontal bone, but inferiorly it extends somewhat over the median line. In form it is about quadrilateral; but it measures two and a half by one and three-quarter inches. Two large pieces of bone are seen to have been detached and upraised, the upper one having been separated at the coronal suture from the parietal bone, and being so closely united that the fracture does not show upon the outer surface. The lower piece shows the line of fracture all around. Owing to the loss of bone, two openings are left in the skull; one that separates the two fragments has nearly a triangular form, extends rather across the median line, and is four inches in circumference; the other, situated between the lower fragment and the left half of the frontal bone, is long and irregularly narrow, and is two and five-eighths inches in circumference. The edges of the fractured bones are smooth, and there is nowhere any new deposit."

From this account it will be seen that the left anterior lobe of the brain suffered severely by this terrible injury, and yet it is not stated that the subject had ever shown any difficulties of speech. If the faculty of language resides in the whole of the lobe, such an immunity could scarcely have existed. It must be noted, however, and the photograph of the cranium establishes the fact, that the third frontal convolution and the island of Reil escaped all injury. Another interesting circumstance is the addiction to profanity after the accident. A like phenomenon has been noticed in cases of aphasia.

---

[1] "Descriptive Catalogue of Warren Anatomical Museum," *loc. cit.*

The second instance is almost as extraordinary. I quote the history of the case, 952, from Dr. Jackson:[1]

"Cast of the head of a man who was transfixed through the head by an iron gas-pipe, and who, to a very considerable extent, recovered from the accident.

"The patient, a healthy and intelligent man, about twenty-seven years of age, was blasting coal when the charge exploded unexpectedly, and the pipe was driven through his head, entering at the junction of the middle and outer thirds of the right supra-orbitary ridge, and emerging near the junction of the left parietal, occipital, and temporal bones. One of his fellow-miners saw him upon his hands and knees, and struggling as if to rise; and, going to his assistance, he placed his knee upon his chest, supported his head with one hand and with the other withdrew the pipe. This last projected about equally from the front and back of the head, and much force was required for its withdrawal."

Brain escaped from the anterior opening, and coma and collapse supervened. "In seven weeks he sat up, and in one more walked about. The right hand he used somewhat, but less well than the left. For about ten months after the accident his memory for some things was nearly lost, but during the next two months there was a considerable improvement."

The accident happened on May 14, 1867, and in June, 1868, the patient, with the gas-pipe, was exhibited to the Massachusetts Medical Society. "The man appeared to be in a good state of general health; and, though his mental powers were considerably impaired, there was nothing unusual in his expression, nor would there be noticed, in a few minutes' conversation with him, any marked deficiency of intellect."

It is very evident that in this case the right anterior lobe was seriously injured—the left escaping—and yet there does not appear to have been any aberration of speech. It is to be regretted, however, that the history is not more specific as to the things in regard to which the memory was deficient.

There are other cases which militate against Bouillaud's doctrine. Thus, M. Peter[2] states that a drunken cavalry-soldier fell from his horse on the back of his head, and fractured his skull. Stupor set in at once, followed by the most violent delirium. The man kept constantly shouting the worst possible oaths, and held connected conversation with imaginary persons. He died at the end of thirty-six hours, without having recovered his reason. On dissection, a fracture of the roof and base of the skull was discovered in all its length. The posterior lobes of the brain were found, on post-mortem examination, to have sustained no injury, but both anterior lobes were in a pulpy condition, through a most violent contusion, caused by their being knocked against the anterior wall of the cranium. The whole thickness of the lobes was disorganized. As Trousseau remarks, this case shows that the two frontal lobes may be destroyed in their anterior portion without causing a loss of the faculty of speech. Trousseau also cites the case of two officers, who, after a quarrel, fought a duel. One of them fired first, and the ball entered his adversary's head at one temple, passed through the brain, and then raised the temporal bone on the opposite side. The ball was extracted, and the patient immediately made a sign with his hands, and expressed his thanks in a very low voice. He recovered, for the time being, and, during five months thereafter, could speak perfectly well, and was remarkable for the wit and fluency of his conversation and writing. He subsequently died of softening; and it was found, on post-mortem examination, that the ball had passed through the two frontal lobes in their middle portion. A still more striking case is referred to by Dr. Bazire, in a note to Trousseau's lecture on aphasia, in the work cited. It was reported in 1843 by M. Aug. Bérard, to the Anatomical Society of Paris. The patient, a miner, was knocked down and severely injured by an explosion in a mine. He did not lose consciousness, but managed to creep out of his hole and to call to his help some men who were working a short distance off. He begged them to fetch a cart and to take him to M. Bérard's house. He was there examined. The whole frontal region was laid open, the integuments hung in shreds, the bones were splintered and in detached fragments, and the brain was exposed. Both anterior cerebral lobes were completely destroyed, and in their stead was a mixture of blood, of bony splinters, and brain-substance. In spite of this frightful injury, the man could relate in all its details how the accident had occurred. He died the next day.

---

[1] *Op. cit.*, p. 149.

[2] Quoted by Trousseau, "Lectures on Clinical Medicine." Translated by Bazire, vol. i., p. 256.

Whether or not we accept this case in all the import claimed for it, there can be no doubt that Bouillaud is wrong in claiming that injury of the anterior lobes is necessarily followed by some derangement in the faculty of speech. It is only fair, however, to state that latterly he has admitted that the organ of language may occupy the posterior part of either lobe.

Dr. M. Dax, in 1836, read a paper before the medical congress which met that year at Montpellier, in which he came to the conclusion that the faculty of language "was seated, not as Gall and Bouillaud had contended, in both anterior lobes of the brain, but that it occupied only the left anterior lobe." He based this opinion on one hundred and forty cases of aphasia attended with paralysis, and in which the loss of power was on the right side; showing, therefore, that the lesion which produced the aberration of speech also caused the hemiplegia, and that this lesion must have been on the left side. This paper at the time attracted very little attention, and was forgotten till the year 1861 witnessed the reopening of the discussion.[1]

It would be very easy to quote a large number of cases confirmatory of Dr. Dax's doctrine, but a few will suffice to show the general bearing of a great many others. The following case seems to have escaped notice. It is not the one referred to by Gall as being sent to him by Larrey. In that case the left anterior lobe was injured and there was aphasia, but the lesion was caused by a sword.

Baron Larrey[2] presented to the Academy the cranium of a subject, with the following history:

Toward the end of the year 1815 an officer of dragoons came to the hospital with a wound from a ball which he had received at Waterloo. The missile had entered the left side of the cranium at a point about six or eight millimetres from the eyebrow and near the temporal ridge. At first he had suffered loss of consciousness and profuse hæmorrhage, but had recovered, with but slight loss of motor power. So far as his mind was concerned, there was no derangement except as regarded the faculty of speech; he had lost the memory of substantives. For this reason he was unable to drill his company, and, though able to distinguish his men by their size, their form, their complexion, or their voice, he could not call them by name. He refused to allow the operation of trephining to be performed, and in 1827 died of phthisis.

A post-mortem examination was made. The ball was found embedded in the thickness of the bone, having elevated and fractured the internal table. The dura mater was strongly adherent to the whole of the left anterior cranial fossa; it was also thicker and denser than in the natural state. A spheroidal excavation, five centimetres in its horizontal and seven or eight in its vertical diameter, was discovered at the summit and on the temporal side of the left anterior lobe of the brain.

Mr. Thomas Hood[3] reported the history of a patient, a sober, intelligent man, sixty years of age, who, on the evening of September 2, 1822, suddenly began to speak incoherently, and became quite unintelligible to those around him. It was discovered that he had forgotten the name of every object in Nature. His recollection of things seemed to be unimpaired, but the names by which men and things were known were entirely obliterated from his mind, or rather he had lost the faculty by which they were called up at the control of the will. He was by no means inattentive, however, to what was going on, and he recognized friends and acquaintances perhaps as quickly as on any former occasion; but their names, or even his own or his wife's name, or the names of any of his domestics, appeared to have no place in his recollection.

---

[1] Dr. Marc Dax's memoir was republished in the *Gazette hebdomadaire*, No. 17, April, 1865.

[2] "Blessure du cerveau avec perte de mémoire des noms substantives," *Journal de physiologie de Magendie*, tome viii., 1828, p. 1.

[3] "Phrenological Transactions." Quoted by George Combe in his "System of Phrenology," Boston, 1834, p. 429.

"On the morning of the 4th of September," says Mr. Hood, "much against the wishes of his family, he put on his clothes and went out to the workshop, and when I made my visit he gave me to understand, by a variety of signs, that he was perfectly well in every respect, with the exception of some slight sensations referable to the eyes and eyebrows. I prevailed on him with some difficulty to submit to the reapplication of leeches, and to allow a blister to be placed over the left temple. He was now so well in bodily health that he would not be confined to the house, and his judgment, in so far as I could form an estimate of it, was unimpaired, but his memory of words was so much a blank, that the monosyllables of affirmation and negation seemed to be the only two words in the language the use and significance of which he never entirely forgot. He comprehended distinctly every word which was spoken or addressed to him; and, though he had ideas adequate to form a full reply, the words by which these ideas are expressed seemed to have been entirely obliterated from his mind. By way of experiment I would sometimes mention to him the name of a person or thing, his own name for example, or the name of some one of his domestics, when he would repeat it after me distinctly once or twice; but generally before he could do so a third time the word was gone from him as completely as if he had never heard it pronounced. When any person read to him from a book, he had no difficulty in perceiving the meaning of the passage, but he could not himself then read, and the reason seemed to be that he had forgotten the elements of written language, viz., the names of the letters of the alphabet. In the course of a short time he became very expert in the use of signs, and his convalescence was marked by his imperceptibly acquiring some general terms which were with him, at first, of very extensive and varied application. In the progress of his recovery, time and space came both under the general application of time. All future events and objects before him were, as he expressed it, '*next time;*' but past events and objects behind him were designated '*last time.*' One day, being asked his age, he made me to understand that he could not tell; but, pointing to his wife, uttered the words, '*many times*' repeatedly, as much as to say that he had often told her his age. When she answered sixty, he answered in the affirmative."

On the 10th of January he suddenly became paralytic on the left side [this is evidently a typographical error for right side]. On the 17th of August he had an attack of apoplexy, and on the 21st he expired. In the *Phrenological Journal*, vol. iii., p. 28, Mr. Hood has reported the dissection of his brain: "In the left hemisphere, lesion of the parts was found, which terminated at half an inch from the surface of the brain, where it rests on the middle of the supra-orbital plate." Two small depressions or cysts were found in the substance of the brain, "and the cavity considered as a whole expanded from the anterior part of the brain till it opened into the ventricle in the form of a trumpet. The right hemisphere did not present any remarkable appearance."

Dr. Thomas Hun,[1] of Albany, in detailing a case of amnesia in which there were no symptoms of paralysis, and in which there was no post-mortem examination, cites the case of a lady who died of cancer of the brain, occupying, at the time of her death, the greater portion of the left anterior lobe. In the early stages of her disease she was often unable to call the most familiar objects by name, and had to express herself by signs or by pointing at the object. When the word she wanted was pronounced before her, she recognized it, and was able to repeat it.

Other cases, and especially several which have occurred in my own experience, are reserved for future consideration.

Up to this period we have the organ of articulate language limited to the left anterior lobe of the brain, but in 1861 its location was still further restricted. In that year M. Gratiolet, in discussing before the Anthropological Society of Paris a question relative to the comparative development of the brain and mind among different races, brought up the subject of cerebral localization, to which he announced himself as being strongly opposed. M. Auburtin, on the contrary, contended that the localization of the faculty of speech at least was definitely established, through the researches of Bouillaud, in the anterior lobes. In support of this view, he adduced cases which had already been brought forward, and cited others in addition, which went to show that loss of speech was the consequence of traumatic lesion of these parts of the brain. His adversaries cited other cases in which persons had preserved the faculty of language notwithstanding extensive lesions of the anterior lobes. M. Auburtin responded that, if such profound and extensive injuries had not interfered with speech, it was because that part of the lobes in which the organ is situated was not involved. And he then cited the case of a patient in the Hospital for Incurables, who for many years had been deprived of the power of speech, and he declared that he would renounce the doctrine of Bouillaud if the autopsy of this patient did not reveal disease of the anterior lobes. The patient in question was under the charge of M. Broca, and the latter, a decided opponent, accepted the challenge of M. Auburtin, and declared that, when the man died, the examination should be made.

Some time afterward the patient died, the post-mortem examination was made, and the lesion was found to occupy the left anterior lobe.[2]

From this time forward, M. Broca, who had been a most determined opponent of Bouillaud's views of localization, became converted, and carried them to a still more extreme point than even M. Marc Dax had done. Taking, as his principal case, the one to which M. Auburtin had pinned his faith, he read, in 1861, before the Anatomical Society of Paris, a memoir,[1] in which he discusses the question of the location of the faculty in question with all his perspicuity and directness. As the two cases cited by him are of historical interest, I give the chief details of them:

---

[1] *American Journal of Insanity*, vol. vii., 1850–'51, p. 359.

[2] See "Étude sur la localisation de la faculté du langage articulé." Thèse de Paris de M. Carrier, 1867.

A man named Le Borgne, who had been an inmate of another department of Bicêtre for over twenty years, was transferred to one of the wards under M. Broca's care, to be treated for a severe attack of phlegmonous erysipelas. The man was a confirmed epileptic, and had not spoken, since his entrance into the hospital, more than a few words, which he employed for the expression of all his ideas. It is stated that in other respects his intelligence was good. Le Borgne was known in the hospital by the name of "Tan," a word which he habitually used, and which, with the oath, "*Sacré nom de Dieu*," constituted his entire vocabulary. "Tan," owing to the constancy with which he used it, was the name by which he was known in the hospital; and, when he could not make himself understood by his signs, he employed the oath, and gave other manifestations of anger.

For several years he had remained in the hospital with no other lesion than that of speech, with an occasional epileptic paroxysm; but, after a few years, his right arm became paralyzed, and four years subsequently the leg of the same side was involved; his sight was likewise enfeebled, and for the past seven years he had been entirely confined to his bed.

Notwithstanding the fact that he was almost in a dying condition when M. Broca first saw him, some important points in his cerebral difficulty were noted. To any question put to him, he replied, as usual, "*Tan*," but at the same time endeavored to make himself understood by signs. Thus he raised six fingers to indicate that six days had elapsed since the inception of his erysipelas, and by opening and shutting his hand four times and then raising one finger signified that he had been twenty-one years in Bicêtre.

Sensibility was lessened on the affected side; there was no deviation of the tongue, which could be moved freely in all directions, and no paralysis of the face beyond a slight weakness shown by the swelling of the left side when he breathed; there was a little difficulty of swallowing, from the fact that the muscles of the pharynx were gradually becoming implicated. After a few days the man died.

As I have said, the autopsy showed that the lesion was situated in the left anterior lobe. More exactly, however, it should now be stated that it involved the inferior marginal convolution of the temporo-sphenoidal lobe, the convolutions of the island of Reil, and in the frontal lobe, the frontal transverse convolution, and the posterior half of the second and third frontal convolutions. The left corpus striatum was also affected. According to Broca, the disease had in all probability begun in the third frontal convolution, and had gradually extended to the other parts; the paralysis marking the implication of the island of Reil and the corpus striatum.

---

[1] "Sur le siége de la faculté de langage articulé avec deux observations d'aphemie." *Bulletin de la société anatomique*, tome iv., 1861.

The other case was that of a man named Le Long, aged eighty-four years, who had entered the hospital for a fracture of the neck of the femur. Eighteen months before, he had been treated in the medical service for a temporary apoplexy, which had deprived him of the faculty of speech, but had caused no paralysis. Le Long, whose intelligence, facial expression, and ability to gesticulate, were very striking, made himself perfectly well understood, although able to pronounce indistinctly a very few words, but which were nevertheless properly applied. These words were "*oui*," "*non*, *toujours*, *tois*" for *trois*, and *Lelo* for *Le Long*. Thus when asked, "Can you write?" he answered, "Oui." "Have you any children?" "Oui." "How many?" "Tois," but at the same time, as if aware that he was not answering correctly, he raised four fingers. "How many boys?" "Tois," raising two fingers. "How many girls?" "Tois," holding up two fingers. "What time is it by this watch?" "Tois," at the same time raising ten fingers to signify that it was ten o'clock. "How old are you?" To this question he replied by two gestures; the one consisting of raising eight fingers, the other of four fingers, by which he meant that he was eighty-four years old.

Aside from this application of the word *tois* to all numbers, his answers were perfectly correct. The tongue was neither paralyzed nor thickened; on one side the larynx was mobile, and his limbs possessed their normal power for his age. It was therefore a case of pure aphasia, or, as Broca then designated the affection, aphemia.

Twelve days after the accident, the patient died. The post-mortem examination revealed the existence of lesions, almost identical in situation with those of the former case. The posterior part of the third left frontal convolution, and the contiguous part of the second, had been absorbed and replaced by a serous fluid. Two cases can scarcely decide any point in pathology; but, without venturing to assert positively that the organ of language resides exclusively in the posterior part of the third frontal convolution, M. Broca expressed the opinion that the integrity of this convolution, and perhaps of the second, is indispensable to the normal operation of the function of speech.

Many cases were adduced by Charcot,[1] by Falret,[2] by Perroud[3] of

---

[1] *Gazette hebdomadaire*, 1863, pp. 473, 525.

[2] *Archives de médecine*, tome iv., Mars et Mai, 1864.

[3] *Journal de médecine de Lyon*, Janvier et Février, 1864.

Lyons, by Trousseau,[1] and others, in support of the localization of the faculty of articulate language in the left side of the brain. Most of these cases were accompanied by right hemiplegia, and, in several, post-mortem examinations showed the lesion to exist in the parts designated by Broca.

In the early part of 1833, M. G. Dax, son of the M. Dax who had placed the organ of language in the left hemisphere, presented, through M. Lelut, a memoir to the Academy, in which he claimed with his father that aphasia was always the result of lesion of the left hemisphere, but he assigned a still more restricted position, by limiting it to the anterior and exterior part of the middle lobe. He cited forty cases of loss of the power of speech, coincident with lesion of the left hemisphere.

Now, besides these direct cases, there are others which bear with almost as much effect on the affirmative of the doctrine in question. Thus M. Fernet, in 1863, presented a case to the Société de Biologie, in which there was left hemiplegia, but no aphasia. After death, softening of the right hemisphere, from thrombosis of the right middle cerebral artery, was found to exist. M. Parrot[2] adduced another case in which there was complete atrophy of the island of Reil, and of the third convolution of the right side, but in which there was no trouble of speech. These cases go to show that the organ of articulate language is not situated in the right hemisphere.

M. Lesur[3] has reported a case which is of very great interest. A child was kicked on the head by a horse, and a fracture of the frontal bone was thus produced. The operation of trephining was performed at a point about an inch and a quarter above the left eye. After the operation and during the progress of the case, it was observed that, whenever pressure was made upon the brain through the hole in the cranium, the child lost the power of speech, and that when this pressure was removed she regained it. A similar case occurred several years ago in my own practice.

Among British writers, Dr. Hughlings Jackson[4] has given the histories of thirty-four cases of loss of speech coinciding with right hemiplegia. He is entitled to the credit of making a beautiful application of anatomy and physiology to the pathology of the subject under consideration. The part of the brain designated by Broca as the seat of the organ of articulate language is nourished by the left middle cerebral artery. An obstruction of this artery would of course interfere with the perfect action of that region, and thus aberrations of speech would be produced. But the same artery also supplies blood to the corpus striatum of the same side. Hence the frequency with which aphasia is associated with right hemiplegia. The cause of the obstruction is generally, according to Dr. Jackson, embolism, for in twenty of his cases the heart was more or less affected, and in thirteen of them there was valvular disease.

---

[1] *Clinique médicale.*

[2] *Gazette hebdomadaire*, 1863, p. 506.

[3] *Gazette des hôpitaux.*

[4] "London Hospital Reports," vol. i.

Among other British writers, some of whom will be more fully referred to hereafter, must be mentioned, Dr. Sanders,[1] Dr. Moxon,[2] Dr. Ogle,[3] Dr. Bateman,[4] and Dr. Bastian.[5]

The matter does not appear to have attracted much attention from German physiologists and pathologists, since the discussion in the French Academy in 1861. Previous to that period several excellent memoirs upon the physiology of speech were published by Germans, among which that of Dr. Bergman[6] is preëminent. A memoir by Nasse[7] is also interesting.

In 1865 Von Benedict and Braunwart[8] published a very thorough paper on the subject, and other observers have reported cases.

In this country there have been several very excellent memoirs upon aphasia, and, as we have already seen, the subject early attracted attention, and the fact that such a condition could exist without other manifest symptoms was fully recognized. Thus Prof. A. Flint[9] detailed the histories of six cases, in one of which post-mortem examination showed extensive disease of the left anterior lobe, and in four, in which the situation of the hemiplegia was noted, the right was the affected side.

Dr. H. B. Wilbur,[10] in a memoir on aphasia, treats of the aberrations of the faculty of language as they existed in certain idiots under his observation. His cases, though interesting, are scarcely in point, as the difficulties of speech were clearly the result of mental deficiencies.

A very important memoir is that of Dr. E. C. Seguin,[11] in which a very excellent history of the subject is given, with the citation of forty-eight cases from the records of the New York Hospital, in which there were difficulties of speech coexisting with hemiplegia, and two in which there was no hemiplegia. In several of these cases, however, as Dr. Seguin states, the loss of the faculty of speech was due to paralysis of the tongue and other muscles concerned in articulation.

Another excellent paper is by Dr. T. W. Fisher,[12] of Boston. Dr Fisher has studied the subject very philosophically, and records thirty eight cases in which post-mortem examinations were made with definite results. Cases have also been published by Bartholow[1] and others.

---

[1] *Edinburgh Medical Journal*, August, 1866.

[2] *British and Foreign Medico-Chirurgical Review*, April, 1866.

[3] "St. George's Hospital Reports," vol. ii., 1867.

[4] *Journal of Mental Science*, January, 1868, and subsequent numbers.

[5] *British and Foreign Medico-Chirurgical Review*, January and April, 1869.

[6] "Einige Bemurkungen über Störungen des Gedächtniss und der Sprache. *Allgemeine Zeitschrift für Psychiatrie*, 1849, s. 657.

[7] *Allgemeine Zeitschrift* u. s. w., 1853, s. 523.

[8] Canstatt's "Jahresbericht," 1865, s. 31.

[9] *Medical Record* (New York), March 1, 1866.

[10] *American Journal of Insanity*, July, 1867.

[11] *Quarterly Journal of Psychological Medicine*, etc., January, 1868.

[12] *Boston Medical and Surgical Journal*, September 1, 1870, and subsequent numbers.

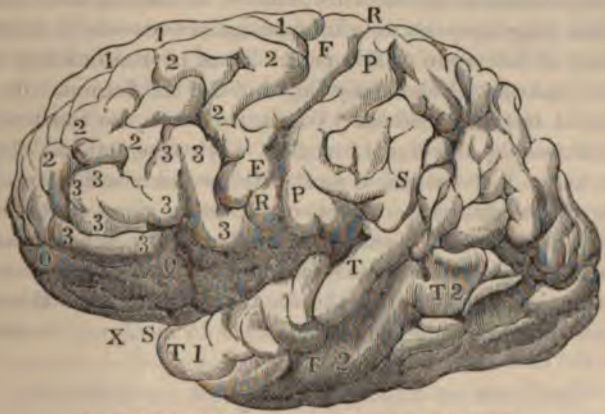
With this outline statement of the history of the subject of aphasia, we are in a position to inquire more fully into the evidence which locates the organ of language in a particular region of the brain.

A clear idea of the anatomy of the parts fixed upon latterly as the seat of the faculty will aid in the understanding of the subject.

The following account is condensed by Dr. Bateman[2] from Broca's description in his essay "Sur le siége de la faculté du langage articulé:"

"The anterior lobe of the brain comprises all that part of the hemisphere situated above the fissure of Sylvius, which separates it from the temporo-sphenoidal lobe and in front of the furrow of Rolando which divides it from the parietal lobe. The furrow of Rolando separates the frontal from the parietal lobe; it traverses from above downward all the external surface of the cerebral hemisphere, starting from the inter-hemispheric median fissure, and ending at the fissure of Sylvius. In front, this furrow is bounded by the transverse frontal convolution, and behind by the transverse parietal convolution. The anterior lobe is composed of two stories or divisions—one inferior or orbital, the other superior—situated beneath the frontal and under the most anterior part of the parietal. This superior division of the anterior lobe is composed of four fundamental convolutions; one posterior, the others anterior. The posterior is that which has been described as the *transverse frontal*,

Fig. 18.



FROM BROCA, AS MODIFIED BY DR. HUGHLINGS JACKSON.

1. First Frontal Convolution; 2. Second Frontal Convolution; 3. Third Frontal Convolution; O. Orbital Convolutions; E F. Transverse Frontal Convolution; P. Parietal Lobe; T S. Temporo-sphenoidal Lobe; T 1. First Temporo-sphenoidal Convolution; T 2. Second Temporo-sphenoidal Convolution; I. Island of Reil; R R. Furrow of Rolando; S. Fissure of Sylvius.

---

[1] *Medical Repertory*, Cincinnati, January, 1869.

[2] *Op. cit.*, p. 522.

and which forms the anterior border of the furrow of Rolando; the three other convolutions have all an antero-posterior direction and are distinguished by the names of *superior or first frontal, middle or second, and inferior or third frontal* convolutions. This last, by its posterior half, forms the superior border of the fissure of Sylvius, the inferior border being formed by the superior convolution of the temporo-sphenoidal lobe. In drawing asunder these two convolutions which bound the fissure of Sylvius, the lobe of the insula (the island of Reil) is exposed, which covers the extra-ventricular nucleus of the corpus striatum. The result of these relations is that a lesion, which is propagated from the frontal to the temporo-sphenoidal lobe, or *vice versa*, will pass almost necessarily by the lobe of the insula, and thence, in all probability, it will extend to the extra-ventricular nucleus of the corpus striatum, seeing that the proper substance of the insula, which separates the nucleus from the surface of the brain, is composed only of a very thin layer."

The lobe of the insula, or the island of Reil, is found in no other mammal than man and the monkey. In the latter, however, it is very slightly developed, and has no trace of convolutions. In aberrations of speech this part is very often involved in the lesion.

Now, although there are several cases on record in which post-mortem examination would appear to show that lesion of the third left frontal convolution is sufficient to produce derangement of the faculty of articulate language, the weight of evidence is decidedly against limiting the seat of the organ to this part. Thus, of five hundred and fifty-six cases of aphasia tabulated by Seguin,[1] the third frontal convolution was damaged but in nineteen. While, therefore, we must admit that injury or disease of this limited region will cause aphasia, it is going too far to assert that the lesion must exist in this situation in order that aphasia may be produced. Moreover, Seguin gives another table of cases which must definitely settle the matter, and which I quote in full. It relates to autopsies which were made with special reference to the point in question, and in which the details given were sufficient clearly to indicate the location of the lesion.

QUESTION OF THIRD LEFT FRONTAL CONVOLUTION.

AUTHORITIES.	For.	Against.
Trousseau, 1865 (in Académie de Médecine)............	14	18
Peter, Legrand, Béclard, Delpech, Bérard, Farge, Jackson, Bigelow..	..	8
Jackson, Richardson, Russel........................	3	..
New York Hospital, 1830–'67........................	1	7
Bellevue Hospital, October, 1867.....................	..	1
Total....................................	18	34

---

[1] *Op. cit.*, p. 97.

Other cases might readily be adduced, but the above are amply sufficient to decide the question against Broca's doctrine. One case of aphasia occurring without lesion of the third frontal convolution would of course invalidate his claim that this part is the exclusive seat of the organ of language, and no number of cases showing coexistence of aphasia with disease or injury of the third left frontal convolution would be sufficient to establish the point affirmatively unless the results of our present experience were disproved. Nevertheless, as showing further that disease of this part will cause aphasia, I subjoin the following case from Dr. W. Ogle's[1] very interesting memoir:

"Joel B., October 18, 1866. Had rheumatic fever and endocarditis twenty-five years ago, but since that has had good health. While at work, October 15th, fell down suddenly without losing consciousness, and found that he was speechless, and hemiplegic on the right side.

"On admission he was found to have extensive heart-disease, with the pulse characteristics of aortic regurgitation. There was complete lax palsy of the right arm and leg, with unimpaired sensibility. There was at first some difficulty in deglutition and in protruding the tongue, but this latter symptom passed away in a few days. There was slight pain in the left side of the head.

"His speech was limited to the two words 'yes' and 'no.' These he used correctly. After he had been in the hospital some time, he recovered the power of saying some few words, chiefly monosyllables.

"He could write with his left hand, with sufficient distinctness, words which he could not pronounce when asked to do so. In his writing there was often a tendency to reduplication of letters. For instance, he wrote 'Testatament' for 'Testament.' But I cannot say whether this was more than the result of deficient education.

"His mind seemed quite clear. He understood all that was said to him; took interest in all that was going on about him; listened to conversation with an animated, lively look, laughing at any little joke, and expressing himself frequently by suitable pantomime. In December he was attacked by œdema of the lungs, and died on the 20th."

Post-mortem.—Œdematous lungs, extensive aortic and mitral disease. "Much semi-gelatinous fluid in subarachnoid space. Surface of brain healthy, excepting at one limited spot. This was the posterior part of the third frontal convolution on the left side. Here was a softened, almost diffluent patch about three-quarters of an inch in breadth, reaching from the highest point of the third convolution backward and downward to the fissure of Sylvius. The softened patch was not actually the most posterior part of the convolution, for there was a narrow unsoftened strip between it and the transverse frontal convolution. In cutting into the brain, a second small patch of softening was seen in the centre of the left hemisphere, external to and rather above the corpus striatum, and extending toward the posterior termination of the fissure of Sylvius. All the rest of the brain was apparently healthy.

---

[1] "Aphasia and Agraphia," "St. George's Hospital Reports," vol. ii., 1867, p. 105.

"The left middle cerebral artery was firm in its main trunk, but in one of its secondary branches at a bifurcation was a hard shotty bit of fibrine completely obstructing the passage, so that when water was injected into the vessel it could not pass, though considerable force was used. There were also fibrinous blocks in the spleen."

The theory of M. Marc Dax locates the faculty of speech in the left hemisphere. He based this opinion upon the fact that aphasia is associated almost, if not invariably, with right hemiplegia, when there is any paralysis at all. That this is really the case is beyond question. Without, however, referring again to the cases cited by M. Dax, I quote the following table from Dr. Seguin's paper:

APHASIA WITH HEMIPLEGIA.

AUTHORITIES.	Right Hemiplegia.	Left Hemiplegia.
Trousseau, 1865 (Académie de Médecine)	125	10
Baillarger, later in 1865 (Salpêtrière)	30	1
Jackson, loc. cit.	34	3
Robertson, loc. cit.	3	..
Medical Times and Gazette, September 9, 1865	2	..
Archives Généraux de Médecine, 1866	2	..
Flint, New York Medical Record, vol. i.	4	..
New York Hospital, 1830–'67	43	3
Total	243	17

From this table we learn that, of two hundred and sixty cases of aphasia associated with paralysis, the left hemisphere—as determined by the situation of the hemiplegia—was the seat of the lesion in two hundred and forty-three cases, and the right in only seventeen.

I also quote the following table from Dr. Seguin:

QUESTION OF LEFT ANTERIOR LOBE.

AUTOPSIES BY	For.	Against.
Marc Dax, in 1861, and G. Dax (Académie de Médecine, 1863)	370	..
Bouillaud, 1848	85	..
" 1865	31	..
Trousseau (Académie de Médecine)	18	16
Vulpian (Leçons de Physiologie)	5	..
New York Hospital, 1830–'67	2	6
Jackson, Richardson, A. Clark, 1866–'67	3	..
Peter, Legrand, Béclard, Delpech, Bérard, one each	..	5
Farge, Bigelow, Detmold, and Stokes, one each	..	4
Total	514	31

This table is based on autopsies, and may be considered conclusi as to the relative frequency with which aphasia is connected with disease of the left anterior lobe.

From various sources I have obtained the following additional cases, in which the seat of the lesion was determined either by post-mortem examination or by the situation of the hemiplegia:

AUTHORITIES.	Left Hemisphere.	Right Hemisphere.
Larrey	1	..
Falret	2	..
Perroud	6	..
Magnan	30	..
Carrier	15	..
W. Ogle	25	..
Bartholow	1	..
Bateman	..	1
W. Wadham	..	1
Total	80	2

The immense preponderance of disease of the left hemisphere, and especially of its anterior lobe, as a concomitant of aphasia, is therefore placed beyond a doubt. Indeed, so far as I am aware, the fact is not questioned. How, now, is it to be explained?

We cannot claim, even with all the disparity of cases, that the organ of language is located in the left anterior lobe, or even in the left hemisphere, to the exclusion of the other. Broca has attempted to account for the assumed restriction, on the ground that the left hemisphere receives a larger supply of blood, and is earlier developed than the right. This is doubtless correct, but still the fact remains that lesion of the right hemisphere is sometimes followed by aberrations of speech; the left remaining perfectly healthy. One such case—and there are several on record in which the autopsy confirmed the deductions drawn from the symptoms—is sufficient to overturn the theory which restricts the situation to one side of the brain; and one such as that reported by Dr. Simpson,[1] in which there was extensive lesion of the third left frontal convolution in its posterior part, and no epilepsy, paralysis, or aberration of speech, is of course utterly destructive of Broca's views.

The fact that aphasia is more frequently conjoined with right hemiplegia is undoubtedly due mainly to the fact previously insisted upon in my remarks on cerebral embolism, that the left middle cerebral artery is much more liable to be plugged by an embolus than the right; and it is by embolism that aphasia is generally caused. Dr. Hughlings Jackson[2] has very satisfactorily worked out the relation, and my own experience, presently to be related, abundantly confirms the fact.

At the same time it appears to be clearly shown that the left anterior lobe, or rather, in accordance with Dr. Jackson's views, those parts of the brain nourished by the left middle cerebral artery, are more intimately connected with the faculty of articulate language than any other region of the encephalic mass. It is probably true, as originally advanced by Dr. Moxon,[1] and since urged by Dr. William Ogle,[2] that the organ of speech is to be found in both hemispheres, and that one side is more generally employed than the other, just as we ordinarily give a preference to one eye or one ear or one hand, and that this side is the left. Gratiolet's facts, adopted by Broca to support his view of exclusiveness, will certainly lend force to the argument in favor of preference. This careful anatomist found that the left hemisphere is developed before the right, and that it is better nourished. Both of these circumstances are owing to the greater supply of blood which it receives.

---

[1] *Medical Times and Gazette*, December 21, 1867.

[2] "London Hospital Reports," vol. i., *loc. cit.*

Undoubtedly many of the cases which have been brought forward as militating against the doctrine of localization of the organ of speech are not cases of aphasia at all, but simply instances of inability to speak, from paralysis of the muscles concerned in speech. This is certainly true of the greater number of Seguin's cases, and also, as Bartholow[3] has stated, of those adduced by Ladame.

Again, in very many instances the post-mortem examination has not been properly made, and lesions involving one or the other anterior lobe have been overlooked. It is now a well-recognized fact that the cerebral tissue may be materially diseased, and the lesion not be detected without microscopical examination.

Giving a very full consideration, therefore, to the facts and arguments which have been urged on all sides of the question, I am constrained, while rejecting the restricted location of MM. Dax, and the still more limited situation contended for by Broca, to believe:

1. That the organ of language is situated in both hemispheres, and in that part which is nourished by the middle cerebral artery.

2. That while the more frequent occurrence of right hemiplegia, in connection with aphasia, is in great part the result of the anatomical arrangement of the arteries which favors embolism on that side, there is strong evidence to show that the left side of the brain is more intimately connected with the faculty of speech than the right.

These views are further supported by several interesting cases, the histories of which I now propose to relate:

CASE I.—In the summer of 1857, while I was on duty, as medical officer of the army, with a body of troops and topographical engineers making a road from Fort Riley to Bridger's Pass, in the Rocky Mountains a quarrel occurred between two of the laborers, which resulted in one of them striking the other a violent blow on the head with a club. The injured man fell to the ground stunned, and remained in a state of coma for several hours. Upon examining him a few minutes after the affair took place, I ascertained that there was no stertor and no indication of paralysis. He was unconscious and breathing quietly, with a pulse of about 80. He had received a blow on the left temple, which, though laying open the scalp, had not fractured the skull. Gradually he regained consciousness so as to be able to comprehend what was passing about him, but he had entirely lost the memory of words, though not the faculty of articulation. Thus he was unable to speak unless the words were first repeated to him, and then he could do so without any defect of articulation, provided too many words were not given to him at once.

---

[1] "On the Connection between Loss of Speech and Paralysis of the Right Side," *British and Foreign Medico-Chirurgical Review*, April, 1866, p. 481.

[2] "Aphasia and Agraphia," "St. George's Hospital Reports," vol. ii., p. 83.

[3] "On Aphasia," *Journal of Psychological Medicine*, etc., vol. ii., p. 341, *et seq.*

Thus when I said to him in Spanish—he was a Mexican, and could not speak English—"Como sientes ahora?" "How do you feel now?" he repeated, "Como sien. sien. sien.," and then, looking at me in apparent despair, burst into tears. And this was repeated time and again during the hour I spent with him.

The next morning, at about seven o'clock, as he attempted to rise from his bed, he fell, and was found a few minutes afterward by the hospital attendant, lying on the ground in a state of complete coma. I saw him almost immediately; he was breathing stertorously, blowing out his lips and cheeks at each expiration, and exhibiting a general resolution of all his limbs. He died at about eleven o'clock A. M. that day.

That afternoon I made a post-mortem examination. On removing the calvarium, the first thing that attracted my attention was an ecchymosed spot about the size of a half-dollar-piece, involving the left anterior lobe at its lateral and posterior margin. There was no extensive hæmorrhage at this point. But, on the opposite side, there had been a rupture of the middle meningeal artery, and an immense extravasation of blood which had infiltrated between the lobes of the right hemisphere and collected in the base of the skull. My theory of the case was that the hæmorrhage from the artery had been suddenly stopped during the condition of primary insensibility before any considerable quantity of blood had been effused, and that during the night his heart had recovered its power; and this, with the muscular effort he made in attempting to get out of bed, had dislodged the coagulum, and allowed the hæmorrhage to take place. At that time I attached no especial importance to the injury of the left anterior lobe; but, since the debate in the French Academy in 1861, I have had no doubt that to it the amnesic aphasia was entirely due.

It will be observed that there was no defect of articulation in this case, either from paralysis or incoördination, but that the difficulty was solely as regarded the memory of words.

Case II.—J. H., a captain of a coasting-vessel, consulted me in November, 1864, for a difficulty of speech with which he had been affected for several months. Upon inquiry, I ascertained that one morning early he had been called from his bed upon some duty connected with his vessel; that he had risen rather hastily and gone on deck; that while giving an order he suddenly became very dizzy, and fell, unconscious. He soon regained his senses, but found that he was paralyzed on the right side, and had lost the ability to speak. He soon afterward reached port, and remained at home for three months, during which period the paralysis disappeared almost entirely, and he reacquired the ability to speak. The aphasia was of both the amnesic and ataxic forms. He could neither speak nor write.

He then went to sea again as a passenger to Cuba, and while in Havana had another attack similar to the first, but without paralysis of motion, though there was loss of sensibility on the right side. The memory for words was entirely destroyed, though he could pronounce distinctly any word he was told to say, if he did not allow too long a period to elapse between the direction and the response. About four months after his last seizure he consulted me.

At this time he could say a few words, and he employed them to express all his ideas, assisting himself with very energetic gestures, which, however, were rarely expressive of his thoughts. The words he thus constantly used were "sifi," which signified both yes and no, and "time of day," which he employed when he had any other answer than a simple affirmative or negative to give. Besides these expressions, he had an oath, "Hell to pay!" which he ejaculated whenever he did not succeed in making himself understood, and sometimes without any such exciting cause. These were the only expressions he could originate, but he could pronounce distinctly any word he was told to say, and even as many as three short successive words. When told to write, he took the pen, and, on my telling him to give me his name and address, wrote "Time of day," and then, seeing that that was not the correct answer, immediately followed it with "Hell to pay!" On my remarking to him that he had given me wrong information, he immediately wrote "sifi." Any word, however, which I told him to write, he did without any difficulty, and thus I obtained several long sentences from him.

From his brother, who came with him, I obtained the facts in his history I have mentioned. Examining his heart, I found that he had a strong systolic murmur, and was told by his brother that he had had, fifteen years ago, a first attack of acute articular rheumatism, which had been followed by several other attacks.

The main point of interest about this case—and it is one of those I shall again draw attention to—is, the occurrence of ataxic aphasia with hemiplegia as concomitants of the first attack, while the second was characterized by purely amnesic aphasia and no paralysis.

CASE III.—During the winter of 1868–'69, a man came to my clinique, at the Bellevue Hospital Medical College, who was aphasic, and from whose friends, his own gestures, and the few words he could speak, I obtained the following history: Some months previously he had been working in a stone-quarry, and was struck by some piece of machinery on the left side of the head, at about the junction of the frontal with the temporal bone. For a short time he was unconscious, recovering, however, without paralysis, but with loss of the memory of words. When he came under my observation, he was very intelligent, comprehended every word said to him, and made repeated and persistent efforts to talk, but he could not utter a word spontaneously beyond "yes" and "no," which he always used correctly. Thus, when I asked him where he was born, he became much excited, gesticulated violently, and apparently made every effort to tell me. The perspiration stood out in large drops on his forehead, but no sound came from his lips. Then the following conversation took place:

"Were you born in Prussia?" "No."

"In Bavaria?" "No."

"In Austria?" "No."

"In Switzerland?" "Yes, yes, yes, Switzerland, Switzerland," at the same time laughing, and moving his hands actively in all directions. He could pronounce words well, but could not write.

I took occasion to speak at length on the subject of aphasia, and gave it as my opinion that there had been a fracture of the internal table of the skull, and that a fragment of bone was pressing on the posterior and lateral part of the anterior lobe. My friend Prof. Sayre was present, and I advised him to trephine the patient, with the view of elevating any depressed piece of bone, and restoring the normal function of that part of the brain. The operation was performed a few days afterward, the patient being placed under the influence of ether. The internal table was found to be fractured, and a splinter was pressing on the posterior frontal convolution. It was removed, and, as soon as the patient emerged from the anæsthetic condition, he spoke perfectly well.

This, as will be seen, was also a case of amnesic aphasia, unaccompanied by paralysis.

CASE IV.—A. E., formerly a bookseller, consulted me in the autumn of 1869 for what was considered by his friends to be, and what probably was, softening of the brain. Before any symptom of disease appeared, he had been noted for his remarkable memory, but was now excedingly forgetful, especially as regarded words. Thus he had forgotten his first name, and could not tell me the names of his children. His conversation was marked by great hesitancy, from his not remembering the words he wished to use, and there was, besides, marked difficulty of articulation, and some words he could not pronounce at all. There was right hemiplegia, which had gradually been getting worse, and which, when I saw him, was extensive enough to interfere materially with the movements of his arm and leg. The left side was not affected, and the tongue and face were apparently not paralyzed. He was subsequently lost at sea in the steamer City of Boston.

This case, therefore, exhibited both the amnesic and ataxic forms of aphasia, and was accompanied by right hemiplegia. I regard the condition as being due to thrombosis, probably of the left middle cerebral artery.

CASE V.—W. W., aged forty-one, entered the New York State Hospital for Diseases of the Nervous System, August 22, 1870, hemiplegic on the right side, and affected with ataxic aphasia. In the month of March, 1868, as ascertained by Dr. Cross, the resident-physician of the hospital, he was seized with a dull pain in the right knee, accompanied with numbness, formication, and pricking sensations, limited to the right foot, while general numbness of the whole side soon supervened. These, with loss of power, gradually extended and increased till at the end of two weeks the patient was entirely hemiplegic. There was at no time any loss of consciousness nor any mental aberration. On the 11th of May following, the patient suddenly lost the power of speech, but his mind remained perfectly clear, and, though he could not utter a word, he understood well every thing that was said to him. He remained nearly completely aphasic for four months, being only able during that time to utter a few sounds, which could not be interpreted into intelligible words.

About September, 1868, he began to enunciate a few words, at first very slowly and indistinctly, and gradually acquired more facility. When I presented him before the class at the Bellevue Hospital Medical College, in November, 1870, although he could talk, his power of co-ordination was very imperfect, and many words were articulated with great difficulty. This trouble was chiefly manifested in regard to labials and linguals, such words as "truly rural," "Peter Piper," "baker," and others of the kind, causing him to make repeated efforts before he could even imperfectly pronounce them. There was no paralysis of the tongue, no deviation when it was protruded, and but very slight if any paresis of the orbicularis oris or other facial muscles. The arm and leg on the right side were profoundly paralyzed.

In this case there was no loss of the memory for words, and no difficulty in writing. It was, so far as the aphasia was concerned, entirely ataxic in character, and accompanied by right hemiplegia.

My opinion was, that the symptoms were to be attributed to thrombosis of the left middle cerebral artery.

CASE VI.—R. M., aged twenty-five, noticed one day that his right foot was unusually cold. A few days afterward he had his first attack of hemiplegia of the right side. Suddenly, and without the least warning, except a severe vertigo, he fell, but immediately arose. There was no loss of consciousness, and with assistance he was able to walk to his residence, a short distance off. His face was drawn to the left side, and speech and memory were slightly impaired.

In February, 1869, having recovered motility, he was seized with another attack of right hemiplegia. This time he partially lost consciousness, and his speech again became affected. By April, 1869, he was able to resume his work as a weaver, but his arm was still weak.

In July he had another attack, which was slight.

In May, 1870, he again suddenly became hemiplegic on the right side. There was no loss of consciousness. The face and tongue were affected. With assistance he walked home, and in a week had quite recovered.

In July, 1870, he had his fifth and thus far last attack. While chopping wood he was suddenly seized with a violent pain in the head, followed by vertigo. He fell, but did not lose consciousness. There were right hemiplegia again, difficulty of speech, and dilatation of the left pupil. For five days afterward he was delirious, but finally recovered, with loss of power in the right arm and leg, and increased difficulty of speech. September 1st, he was admitted to the New York State Hospital for Diseases of the Nervous System. At this time the paralysis had entirely disappeared; the tongue could be moved freely in any direction, and his articulation was perfect. But his memory for words was greatly impaired, though facts and circumstances were remembered perfectly well. His speech was therefore hesitating, and if asked to repeat a sentence of three or four words he could not do it. Thus he could not repeat the words "sugar, coffee, crackers," although he began immediately after I had finished saying them.

Examination showed that the patient had hypertrophy of the heart, with aortic insufficiency.[1] My diagnosis was, repeated attacks of embolism of the left middle cerebral artery, or its branches.

This case was one of partial amnesic aphasia, with ataxic aphasia, which had disappeared with the hemiplegia.

Case VII.—Mrs. S. H. W., aged thirty-two, married. On the 26th of June, 1860, about three weeks after the birth of her child, she was suddenly seized with a severe pain in the right shoulder, which extended down the arm. Symptoms of albuminuria, accompanied by general dropsy, immediately ensued, and in a few weeks the dyspnœa from hydrothorax was alarming. Coma and a convulsion followed. Soon after the fit, which marked the height of her disease, as she was sitting by the bed, resting her head on her folded arms, her right side became completely paralyzed, and she lost the ability to speak. She was not entirely clear in her mind for a week after the attack, but gradually the dropsy disappeared, her intellect improved, and the paralysis became less.

---

[1] I have condensed the histories of this and the preceding case from the reports of Dr. Cross, in my clinical lecture on "Partial Cerebral Anæmia," published in the *Journal of Psychological Medicine* for January, 1871.

At the time of the seizure, the face was drawn to the right side, the tongue deviated in the same direction, and there were strabismus and partial ptosis and paralysis of the orbicularis palpebrarum muscle on the right side. Motility and sensibility in the right arm and leg were, at first, completely abolished, but at the end of ten days she was able to move about, by holding on to a chair. During three years she continued to improve as regarded the paralysis, but for all that period did not speak a word. In the summer of 1863 she became able to say the word "no," and a few months later she could say "yes."

At my request, she allowed me to present her before the class of the Bellevue Hospital Medical College, in November, 1870, on the occasion of a clinical lecture on aphasia.

She was then, and is now, enjoying good health, with the exception of frequent headache. Her countenance is remarkably bright and cheerful, and her whole expression is exceedingly intelligent. She comprehends every word that is said to her, and attends to all her household duties. Yet she is unable to utter any words but "no," "yes," and "dado." The latter is seldom employed, but in her vocabulary signifies affirmation. She uses "yes" for affirmation, and "no" for negation, and both for doubtful or indifferent conditions. Thus, if asked how she is, she answers "Yes, yes, no, no," which means that she is tolerably well. Sometimes she employs these words quite indiscriminately. If asked what that is, pointing to a fan, she cannot tell, nor can she repeat the word fan. She shows, however, that she knows, by making the gesture of fanning herself. She can neither read nor write, although on one occasion she succeeded, after great difficulty, in writing "no." Not long since she suddenly ejaculated, "I don't know!" and a few days ago exclaimed, "How do you do?" but she was not able to repeat either of these phrases, nor did she appear to be aware that she had said them. Her gestures are very intelligent and expressive. The right arm and leg are weaker than on the left side, and the sensibility is less.

There is a murmur at the apex of the heart with the first sound.

Ophthalmoscopic examination showed the vessels of the retina of the left eye to be much larger than those of the right.

In this case I diagnosticated embolism of the left middle cerebral artery.

The aphasia was of both the amnesic and ataxic forms, and was accompanied by right hemiplegia.

CASE VIII.—Mr. B. consulted me in November, 1870, for loss of the memory of words, and fullness and pain in the head, with occasional vertigo. Over a year previously, while in the woods of Minnesota buy ing timber, he had suddenly lost consciousness for a few moments, and on recovering found that he had become hemiplegic on the right side, and had lost the power of speech. For a short time he could not utter a word, but gradually the memory of language, and the ability to coördinate the muscles of speech, returned to him, and he could articulate sufficiently well to be understood. For several months, however, his recollection of words was bad.

For some time he had been under the care of Dr. Hale, of Chicago, a homœopathic physician, who advised him to place himself under my charge. When I first saw him, he could talk quite well, but there was still a hesitancy in his speech, and occasionally words were misplaced or miscalled. Articulation was distinct, and the hemiplegia had disappeared. There was pain, almost entirely confined to the left temporal region. There was the history of acute articular rheumatism, and there was aortic insufficiency.

In this case there had been at first amnesic and ataxic aphasia, with right hemiplegia. As the latter disappeared, the ability to coördinate the muscles of speech was increased, until at last articulation became perfect, and only amnesic aphasia remained.

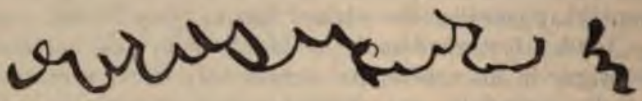
CASE IX.—H. I., a merchant, consulted me in August, 1869, for hemiplegia, with inability to speak. While sitting at his desk, six weeks previously, he suddenly became vertiginous, and lost consciousness for a few moments. On recovering his senses, he discovered that he was paralyzed on the right side, and that he could not speak a word. He was exceedingly anxious to make known some wish, and one of his clerks brought him paper and a pencil, but he could not write a letter. An alphabet was then written, but he was unable to select the letters to form the words he wanted to use.

A physician was sent for, and Mr. I. was bled to the extent of sixteen ounces, without any favorable result. He remained hemiplegic and completely aphasic for about two weeks. He then began to walk, and acquired the ability to say "what," "certainly," and "saw my leg off," which he contracted into "sawmelegoff," accentuating strongly the ultimate syllable. These words he used without apparent intelligence, though he clearly understood all that was said to him, and laughed at any joke as heartily as ever. His condition was about the same when I saw him.

He could protrude his tongue and move it actively in all directions, but could not articulate any words but those mentioned. Thus, when I asked him to say "table," he said "Certainly;" and when I said "Well, say it then," he exclaimed, "Sawmelegoff!" at the same time, to show that he understood what I said, he went across the room, and put his hand on a table, uttering, at the same time, his full stock of words, "what," "certainly," "sawmelegoff."

I then asked him if he could write; he replied, "Certainly." I placed paper before him, and gave him a pen with ink, but he was unable to write his name as I requested, although he could use his fingers for other things tolerably well. I asked him to draw a series of parallel lines, and he did so without difficulty. On my insisting that he should now make an effort to write his name, he made the attempt with this result:

FIG. 19.



I told him that was not his name, at which he gesticulated violently, exclaimed, "Sawmelegoff!" and gave me one of his visiting-cards. This gentleman continued under my care for some time, but with no perceptible change. He had had two attacks of acute articular rheumatism, and had, when I saw him, both aortic and mitral insufficiency.

Here, then, was right hemiplegia, with fully-developed ataxic and amnesic aphasia. My diagnosis was, embolism of the left middle cerebral artery.

CASE X.—Miss C. R., of strongly-marked hysterical diathesis, suddenly became aphasic while sitting at the breakfast-table. I saw her about two hours subsequently, when she drove to my office with her mother. There was no paralysis, the tongue could be moved freely in all directions, articulation was perfect, and she could pronounce any word mentioned before her. The memory of words was, however, entirely abolished.

CASE XI.—Mr. S., a retired merchant, consulted me in September, 1870, for the effects of cerebral hæmorrhage. He was hemiplegic on the right side, and unable to talk. His intelligence was good. He could read, but he was not able voluntarily to pronounce a word. The tongue was not in the least paralyzed, nor had it been. Occasionally ejaculations of various kinds would come forth. On one occasion, as he entered my office, he exclaimed—he was a German gentleman—"Guten Morgen, mein Herr," but by no effort could he repeat that or any other expression. His attempts to speak were continuous while he was with me; and his son who came with him said he was almost always trying to talk while he was not sleeping.

This case was, therefore, one of ataxic aphasia, and was marked by the existence of right hemiplegia. Cerebral hæmorrhage, involving the corpus striatum, was the cause.

CASE XII.—Mr. L. N., a German gentleman, came under my care in September, 1869, for symptoms indicative of cerebral softening. He was slightly paralyzed on the right side. His speech was affected both amnesically and ataxically. Soon afterward, in consequence of maniacal symptoms making their appearance, I sent him, with the concurrence of my friend Prof. Flint, to the Lunatic Asylum, at Flushing. He remained there till September of the present year, gradually failing in mental and physical power, when, as he was no longer in a condition to injure himself or others, his friends, with my approval, removed him to their own home. At the present time he can scarcely remember a word, and his articulation is very defective. A remarkable feature of his conversation is that he calls every thing "kazza," "cat." He appears to have forgotten every other word.

The history of this case points to thrombosis as the probable lesion.

CASE XIII.—This was a very remarkable and instructive case, one which I have already mentioned under the head of embolism.

The patient was a retired officer of the army, and consulted me in the autumn of 1869 for paralysis, vertigo, and slight difficulty of speaking, from which he had suffered for some months. Several years previously he had been under the care of my friend Dr. Metcalfe, for acute rheumatism, with cardiac complications. The history of the case pointed strongly to embolism, and, as the paralysis involved the right side, I diagnosticated a previous attack of embolism of the left middle cerebral artery.

The difficulty of speech was slight; there were both amnesic and ataxic aphasia.

Under the treatment employed he improved very much in the ability to walk, to use his arm, and to speak, so much so that he and his friends considered him better than he had been for several years. But about six weeks after he came under my charge he had another attack. This time the left side was paralyzed, and there was no difficulty of speech. Galvanism was employed, as before, and he recovered sufficiently to go to Washington City. While there he had a third attack, characterized by right hemiplegia and aphasia. He soon recovered his power of speech, and soon afterward had a further attack, involving the left side, and unattended by aphasia. He recovered under the care of Dr. Basil Norris, of the army, and soon afterward came again to New York. A short time after his arrival I requested my friend Prof. Flint to see him in consultation, with the special view of having him examine his heart. This was done with thoroughness, but no abnormal sounds were detected. While in New York he had two other attacks, during both of which he was delirious; both were characterized by hemiplegia. That of the left side was unaccompanied by aberrations of language; that of the right was attended with ataxic and amnesic aphasia. He forgot the names of the most ordinary things, and there were many words that he could not articulate at all. Thus, when he wanted a fan, he called it "a large, flat thing to make a wind with." He forgot my name, and could not pronounce the words beetle, general, physician, and many others. I sent him to Newport greatly improved, but he had other attacks there, and finally died in the autumn of the present year, of, I presume, cerebral softening.

The interesting features of this case are the concurrence of hemiplegia and ataxic and amnesic aphasia, and the striking fact that there was no aphasia when the paralysis involved the left side. Thus, according to my views of the case, the patient had repeated attacks of cerebral embolism. When the embolus lodged in the left middle cerebral artery, there was aphasia accompanied by right hemiplegia; when the embolus obstructed the right middle cerebral artery, there was left hemiplegia, but no aphasia.

CASE XIV.—In the early part of December, 1870, J. M., a patient of Bellevue Hospital, was, at his request, brought to my clinique at the college. His history, as given me by Dr. Judson, showed that he had repeated attacks of unconsciousness or semi-unconsciousness, which were accompanied with hemiplegia. Dr. Flint had also detected a bellows murmur, but it was at the apex of the heart. The patient had suffered from several seizures of acute articular rheumatism.

Upon inquiry, I ascertained that he had had altogether eleven attacks of vertigo, unconsciousness, and hemiplegia. His intelligence was good, and he spoke tolerably well, though with hesitation and occasional difficulty of articulation. His speech was much better than it had been, and there was no well-marked hemiplegia.

As in the case last mentioned, whenever the hemiplegia had been on the left side there was no aphasia, but when it was on the right side there was always well-marked difficulty of speech, both amnesic and ataxic.

The only other case, similar to these last two, that I have been able to find, is one reported by Dr. Stewart,[1] of a man who was admitted into the Middlesex Hospital, suffering from left hemiplegia, without aphasia. A week later he became affected with right hemiplegia and loss of speech. He died, and on post-mortem examination both middle cerebral arteries were found plugged with emboli.

CASE XV.—Captain C., an officer of the mercantile marine, was attacked in September, 1874, with sudden loss of the power of speech, attended with confusion of ideas, and vertigo. He soon recovered, but had several subsequent seizures, characterized by vertigo, impairment of language, and slight delirium. I first saw him on the 31st of October, and on the 28th of November he went with me to the University of New York, where he was one of the subjects of my clinical lecture on aphasia, delivered to the medical class. At this time, and for several weeks previously, he had constantly used words which were without relation to the things he wished to name. Thus, if he wanted his boots, he would ask for his top-sails, or would be apt to employ some other word designating part of a ship. In his conversations with me he continually exhibited this peculiarity. There was no want of memory for any other parts of speech than substantives. For instance, I held up a penknife before him; he at once said it was to cut with, but, when I pressed him to name it, he called it a "boat." A thermometer was an "anchor," and a watch was a "capstan." When I asked him to say *National Intelligencer*, he said "National intelligence-office," and, no matter how often I repeated the words, he always said "National intelligence-office." The reason for this was very obvious: he had frequently had occasion to say "intelligence-office," but had probably never before in his life been asked to say "*National Intelligencer*." After a time, he succeeded in acquiring the power to utter the final "*e r*," but then he placed it in the wrong position, and said "National intelligence-officer." Syllable by syllable, he could speak these words correctly, but they were at once forgotten. This was a typical case of the amnesic form of aphasia.

---

[1] *Medical Times and Gazette*, July 9, 1864.

Many other cases of aphasia have come under my observation, but it is scarcely necessary to mention them in detail, as they present no features differing in any material point from those cited.

The views which the cases I have observed have led me to form, have been confirmed by my recent study of the subject of aphasia. These have already been given in part, but the detail of the foregoing histories enables me to express the remainder with more confidence.

It cannot have failed to strike the reader that, in all the cases of which hemiplegia formed a feature, the aphasia was of the ataxic form, while when there was no hemiplegia the aphasia was amnesic. In the one the individual was deprived of speech, because he could not coördinate the muscles used in articulation, in the other because he had lost the memory of words.

This is a point which has not hitherto been noted. The phenomena indicate, I think, very clearly, the seat of the lesion, and the physiology of the parts involved.

The gray matter of the lobes presides over the *idea* of language, and hence over the memory of words. When it only is involved, there is no hemiplegia, and there is no difficulty of articulation. The trouble is altogether as regards the memory of words.

The corpus striatum contains the fibres which come from the anterior column of the spinal cord, and is besides connected with the hemisphere. A lesion, therefore, of this ganglion, or other part of the motor tract, causes paralysis of motion on the opposite side of the body. The cases I have detailed show, without exception, that the power of coördinating the muscles of speech is directly associated with this hemiplegia. A lesion, therefore, followed by hemiplegia and ataxic aphasia, indicates the motor tract as the seat. If amnesic aphasia is also present, the hemisphere is likewise involved. An analysis of the cases reported by Ogle, Jackson, and some other observers, shows that the association existed in their cases, although they have not noticed it as of any physiological or pathological bearing.

Another important feature of the foregoing cases is the constant association of the aphasia with right hemiplegia where there was any paralysis at all. This indicates, perhaps, only the more frequent occurrence of embolism on the left side, but the last two cases, as well as the one quoted from Dr. Stewart, show that the left hemisphere is more intimately connected with the faculty of speech than the right. In fact, it appears to me impossible to avoid this conclusion.

Prof. Ferrier[1] has quite recently enunciated views relative to the patho-physiology of aphasia, which differ in several respects from those I have just expressed. The importance of the subject, and the deservedly high position in cerebral physiology occupied by their author, must be my apology for quoting them in full:

"Much has been written on the subject of aphasia, and many differences of opinion still continue to exist among those who have directed their attention to it. It is not my intention to enter into any lengthened examination of the theories which have been advanced, nor to controvert the opinions of those who deny all localization of brain-function including a 'speech-centre.' The researches of Broca, and the numerous confirmations of his observations which have been put on record, taken with the results of my experiments on monkeys, and the lower animals, seem to me to establish the fact of a localization of the faculty of speech, and to explain at least the broad features of the pathology of aphasia. I have shown that the region which governs the movements concerned in articulation is that which is the seat of lesion in aphasia. The region is symmetrically situated in both hemispheres, each one possessing the power of originating coördinate movements of the lips and tongue, in a bilateral manner. Broca's convolution is usually described as being the posterior third of the inferior frontal convolution of the left hemisphere. It would be more in accordance with the facts of experiments on monkeys, as well as with the cases which have come under my own observation, to localize the speech-centre in the *operculum* (Klappdeckel), which is included between the ascending and horizontal limbs of the fissure of Sylvius, and which immediately overlaps the island of Reil. While Broca's convolution is placed in the left hemisphere, the centre for bilateral coördination of the speech-muscles exists, as I have said, in both hemispheres. To this bilateral action of each centre is to be ascribed the peculiarity of loss of the faculty of speech without paralysis of the articulating muscles. For, as regards the mere muscular action, one centre is capable of carrying it on when the other has been disorganized. This contrasts in a significant manner with the coincident hemiplegic paralysis of the arm and occasionally of the leg, which frequently exists along with the aphasia. The centres for the movements of the arm and hand are in close contiguity to the centre of articulation, and hence the lesion which causes aphasia usually involves these also. More rarely the leg is affected, owing to the fact that, being at a greater distance, the centres are less often invaded by the softening. These results are to be found when the cortex of the brain alone is implicated, the ganglia being intact, and furnish conclusive proof of the localization in the brain-cortex of the centres for voluntary motion. These centres, however, have another signification, in so far as they form the motor substrata of mind. Besides being centres for the accomplishment of acts of volition, they form the organic centres of the memory of accomplished acts. The centres for articulation, besides their function of setting in action the complex and delicate movements involved in articulate speech, have the power of permanently recording the results of their functional activity. Words represent the movements of articulation, guided by impressions of mind. The memory of words must have organic basis in that part of the brain which is the centre involved in their execution, for the memory of words is nothing else than the memory of the articulating processes, which have been affected under the guidance of auditory sensations. The ideas of which words are the articulatory symbols have no relation to that part of the brain where words are remembered, except by associating fibres. The ideas, in physiological language, have their organic seat in those parts of the brain specially related to the nerves of common and special sensation, for all ideas are ultimately reconcilable to impressions of sense as their basis. The records of these in the brain cells furnish the material of all thought, and by their various combinations and associations constitute the foundation of all complex conceptions. The utterance of a word, with an appreciation of its meaning, involves the physiological activity not merely of the speech-centre, but also of those parts of the brain related to the various senses concerned in the perception of the qualities of the object signified. Thus the mention of the word 'orange' indicates not only the activity of the speech-centre, but also of the sensory centres of color, smell, taste, form, etc., all of which are concerned in our concept of the object. This is a concept of the simplest form, but the analysis of more complex ideas must necessarily be the same in principle.

---

[1] "Pathological Illustrations of Brain Function," "West Riding Lunatic Asylum Medical Reports," London, 1874, vol. iv., p. 54.

"In aphasia consequent, as it usually is, on disease of the left hemisphere, the *memory* of words is not lost, nor is the person incapable of appreciating the meaning of words uttered in his hearing. What is lost in aphasia is, as Hughlings Jackson so clearly discriminates, merely the power of voluntarily using words to express ideas. As both sides of the brain are symmetrical, and work conjointly, the memory of words may remain in the right hemisphere after the occurrence of lesion in the left. The explanation of the loss of the faculty of speech is to be attributed to the unequal preponderance of the left hemisphere in the inclination of voluntary actions. The left hemisphere, like the right side of the body, is the leading or driving side, so that lesion of the left side is like the loss of the right hand. It requires long education to enable the person to accomplish, with his left hand, all the delicate manipulations of which the right hand was capable. The leading action of the left hemisphere may, however, be merely an accident of education or necessity, and there is no reason why articulate speech should not be the function of the right side. That such may be the case there are pathological grounds for believing, viz., in those where aphasia has resulted from disease of the right hemisphere, and in those rare cases where lesion of the left speech-centre has not caused the usual results."

It is very evident, however, that the view of Dr. Hughlings Jackson, thus enunciated and indorsed by Dr. Ferrier, is not of uniform application to the phenomena of aphasia; for, though in some cases there is no loss of the memory of words, but merely a loss of the voluntary power to utter them, there are many other cases in which the patient has lost the memory of words, and has not lost the voluntary power to articulate them. Thus, the person who speaks of his boots as his "top-sails," has not lost the voluntary power of saying "boots," for he can utter the word when asked to do so, an act which would clearly be impossible if he had no voluntary power to say "boots." I have witnessed several cases in which the patients could say any word they were told to say, but who could scarcely articulate a syllable when not prompted. Of these, Case III., described in this chapter, is a remarkable instance, and others will, in this connection, attract the attention of the reader. The fact appears to be, that the only cases in which the voluntary power of articulation is lost, are those which are described in the present chapter as instances of ataxic aphasia.

So much for some of the various theories which exist relative to the localization of the organ of language and for the clinical history of aphasia. I have not thought it necessary to discuss the view of Schroeder van der Kolk,[1] that the faculty of articulate speech resides in the corpora olivaria, because there is little if any physiological or pathological evidence to sustain it. Nor the hypothesis of Brown-Séquard,[2] that speech is a reflex phenomenon, because there is no evidence in support of that opinion. Neither have I, though much tempted, ventured into the philosophy of the subject to any considerable extent.

As to the causes, the prognosis, diagnosis, morbid anatomy, and pathology, they have been sufficiently considered in the remarks made, and the treatment is of course that of the pathological condition to which it is due, whether this be cerebral hæmorrhage, embolism, thrombosis, softening, hysteria, wounds, the bites of poisonous serpents, syphilis, or other cause. One point, however, should be mentioned in this connection, and that is that constant efforts should be made to develop the uninjured speech-centre and to exercise the vocal organs, by constant attempts to speak. The application of the galvanic or faradaic currents to the tongue and other muscles concerned in articulation, is a measure of usefulness.

---

[1] "On the Minute Structure and Functions of the Spinal Cord and Medulla Oblongata" "New Sydenham Society Publications," p. 140.

[2] Seguin's "Memoir,' already quoted.

CHAPTER VIII.

ACUTE CEREBRAL MENINGITIS.

By acute cerebral meningitis is understood inflammation of two membranes of the brain—the pia mater and arachnoid. Some writers have made the attempt to discriminate between inflammation of the arachnoid and inflammation of the pia mater, but there are no diagnostic marks by which such a distinction can be made, and we find from post-mortem examination that neither membrane can be inflamed without the other participating in the morbid process. Inflammation of the dura mater is never included under the term meningitis.

The ancients made no distinction between the several inflammatory affections of the intra-cranial organs, but comprehended them all in one disease, which they called frenzy—*φρήν*, the brain. Morgagni, however, showed that the membranes of the brain were the parts generally involved, and gave a very accurate account of the phenomena of an attack of acute meningitis. Since then, Rostan, Lallemand, Andral, Bouillaud, and others, have added to our knowledge.

Symptoms.—The symptoms of acute cerebral meningitis may be divided into three groups, arranged in chronological order: the stage of invasion, the stage of excitation, and the stage of collapse.

1. The Stage of Invasion.—The most prominent initiatory symptom is headache, which may be diffused or confined to a limited part of the head. When this latter is the case, the frontal region is more generally its seat; next in order of frequency is the occipital, and next the temporal. At the same time the face is flushed, the eyes are red and suffused, and there is a decided elevation in the temperature of the head, which is not only felt by the patient, but may be perceived by the hand of the physician. Vomiting is generally present.

As might be expected, these symptoms are accompanied by fever. This, however, rarely runs high, so far as the force or the frequency of the pulse is concerned, or as regards the heat of the skin. It is mainly characterized by restlessness and insomnia. Occasionally there is a tendency to somnolence.

This stage may last a few days or only a few hours, or may be so slight as not to attract attention. In general features it resembles the prodromatic stage of cerebral congestion.

2. THE STAGE OF EXCITEMENT.—A chill ushers in this stage, and an increase in the intensity of several of the symptoms of the first stage and the development of others soon take place. Thus the fever becomes higher, the skin hotter, and the temperature of the body is elevated several degrees—the thermometer rising as high as 105°, 106°, and sometimes to 107°. The pulse is frequent—rising to 120, or even 160—quick and hard, and the face becomes redder than in the first stage. The pain in the head augments in violence, and is increased by pressure on the scalp, or even the slightest movement.

The eyes are bright, the pupils contracted and painfully sensitive to light. The hearing becomes morbidly acute, loud noises cause great agony, and even slight sounds are unbearable. The general sensibility of the body is increased, and hence the patient is rendered uncomfortable by the contact of the bedclothes with the skin. Delirium is generally present from the first, and is often of furious character. Hallucinations of sight and hearing are almost constant, and the irrationality of the ideas is marked by the incoherence of the speech. The patient when awake is continually talking, gesticulates violently, and weeps and laughs alternately over imaginary evils. It is sometimes necessary to use restraint to prevent him injuring himself or others, and the attendants should always be prepared for any emergency of the kind. As the disease advances, the delirium becomes more subdued, and the patient may exhibit some evidences of sanity.

Even when there is no delirium, as occasionally happens, the influence of the morbid action over the mind is shown in the irritability of the patient, and the change which he undergoes in character and disposition.

Convulsions rarely occur in adults, but motility generally is nevertheless disordered. The limbs are in almost continual action, as are likewise the jaw and the eyelids. Twitchings of the facial and other muscles, such as those of the forearm, are usually well marked, and occasionally there are irregular movements of the eyeballs. Convulsions, when they occur, may be either clonic, or tonic, or both. Thus there may be a gradually-increasing rigidity of some muscles, followed by relaxation and disordered movements. Sometimes there is opisthotonos as well marked as in some cases of tetanus. Hemiplegia or paraplegia may occur, but are infrequent complications. I have seen two cases in which one lateral half of the body was paralyzed during the whole course of the disease.

Contractions of the limbs sometimes take place, and may be confined to one side or a single limb. In this case the forearm is usually strongly flexed on the arm.

The muscles of organic life participate, and the bowels are obstinately constipated. There may be difficulty of swallowing, from spasm of the pharynx, and irregularity of breathing, from implication of the respiratory muscles.

The most characteristic symptom of this stage is, however, the obstinate and violent cephalalgia, of which mention has already been made, and yet there are cases in which it is entirely absent from first to last. Several such instances have been under my own charge, and post-mortem examination has verified the existence of the evidences of meningitis. This stage lasts from a few days to two weeks.

3. THE STAGE OF COLLAPSE.—The beginning of this stage is marked by the occurrence of somnolence, which often shows a tendency to pass into coma, and by a subsidence of the delirium and muscular agitation. There are times, however, during which the stupor remits in profundity, and the patient appears to be somewhat conscious of his condition, but these periods only occur in the first part of the third stage. Ere long the coma becomes constant.

Paralysis then supervenes, and is first manifested in the ocular or facial muscles. Thus from paralysis of one of the muscles of the eyeball strabismus ensues, or the upper eyelid may drop from paralysis of the levator palpebræ superioris. The pupils dilate and become insensible to light, and the mouth is drawn to one side from implication of the muscles of the face. Before long the contractions of the limbs relax, and paralysis takes place. The sphincters of the bladder and rectum also lose their power, and the urine and fæces escape involuntarily. The pulse becomes slow and irregular, but the temperature, as Jaccoud has shown, and as I have lately verified in several instances, does not fall. Some authors regard this reduction in the frequency of the pulse while the heat of the body remains high, as pathognomonic. The insensibility becomes more and more profound, and the patient dies in a state of coma, sometimes from asphyxia produced by paralysis of the respiratory muscles, but generally from the gradual engorgement of the lungs, and with a bodily temperature as high as at any other period of the disease.

Such is the ordinary course of an attack of simple acute cerebral meningitis occurring in a young and healthy person. Though it is certainly true, as post-mortem examinations have shown, that the morbid process may be general or limited to the convex or basilar surface of the brain, or to the ventricular lining, yet during life the distinction cannot be made, mainly for reasons which will be given under the head of pathology. But there are modifications often met with which require consideration. Of these, epidemic cerebro-spinal meningitis, though scarcely to be considered a disease of the nervous system, and tubercular meningitis, will be discussed under other heads, but the differences due to acute rheumatism and old age may very properly be noticed in the present connection.

RHEUMATIC MENINGITIS.

Under the name of cerebral rheumatism, several very different affections of the brain supervening during the course of acute articular rheumatism have been embraced. The relation of rheumatism to such secondary diseases has long been recognized, but very great confusion has existed in regard to the exact nature of the morbid processes set up in the brain and its membranes. That meningitis may, however, be one of these conditions, appears to be quite certain. Gintrac[1] has collected twenty-one cases of cerebral meningitis the result of rheumatism, or at least occurring in conjunction with that disease, the existence of which was established by post-mortem examination. Oulie[2] contributes four others, and many more are to be found in medical treatises and periodicals.

Although I have witnessed a number of cases of what in former editions of this work was designated cerebral rheumatism, I have only had one case in which the existence of meningitis as a consequence of rheumatism was demonstrated by post-mortem examination.

The membranes of the brain are most liable to be affected during the latter stage of an attack of acute rheumatism, but there seems to be no doubt that the cerebral disease in question may supervene at any time during the course of the primary disease, and that it sometimes has all the appearance of being a true metastasis. The symptoms which indicate the supervention of cerebral meningitis are delirium, convulsions, or more frequently choreiform movements in the limbs, tremor, especially about the lips and muscles of the face, paralysis in various parts of the body, and stupor. Pain and vomiting, which are such constant features of ordinary meningitis, are rarely present in the rheumatic form of the affection. The bodily temperature is not elevated more than three or four degrees above the normal standard. Toward the last, coma, if already present, becomes more profound, or if not, makes its appearance, and death ordinarily ensues. Occasionally, however, recovery takes place.[3]

---

[1] *Op. cit.*, tome iii., p. 77.

[2] "Du rheumatisme cérébrale." Thèse de Paris, 1868.

[3] In a very valuable memoir on "Cerebral Rheumatism," just published, Prof. Da Costa[1] has given the details of twelve cases in which cerebral symptoms supervened during the course of articular rheumatism. Dr. Da Costa expresses the opinion that all cases of what is called cerebral rheumatism are not characterized by the presence of meningitis, and the results of the post-mortem examinations which he obtained from his cases, cer-

---

[1] *American Journal of the Medical Sciences*, January, 1875, p. 17.

SENILE MENINGITIS.

In old persons, the symptoms of acute meningitis are rarely so pronounced as in individuals of middle age. The affection comes on more gradually, and may have made considerable progress before its existence is suspected. There is little or no pain, no fever, and no gastric or intestinal derangement. The mental symptoms are very similar to those due to softening. The patient has imperfect articulation, his memory is impaired, and he does things which show that he is not in his right mind. The delirium is of the low muttering kind, and there is a tendency to coma even in the first stage. There is a more or less general paresis in all the limbs, and subsultus is commonly present. Death is usually due to pulmonary engorgement.

Causes.—Among the predisposing causes of acute cerebral meningitis, age is first to be considered. Guérsant[1] asserts that the period of life between sixteen and forty-five is that during which acute meningitis is most liable to occur, not including children, who are far more prone to the disease than adults. Rilliet and Barthez[2] have, however, shown that very young infants are not so subject to simple acute meningitis as children of from five to eleven years of age. The very opposite opinion is expressed by Drs. Meigs and Pepper.[3]

Thirteen cases of acute simple meningitis have come under my observation. Of these, all were between the ages of thirty and forty.

Men are more subject to it than women. Of my cases, ten were males and three females. Parent-Duchatelet and Martinet,[4] however, think women are more predisposed to the affection than men.

Temperature, either very high or very low, predisposes to acute meningitis. Eight of the cases under my care occurred in summer and five in winter.

Certain professions and habitudes appear to favor the occurrence of the disease. Among the former are all those which require the head

---

tainly support this view. But in Case I.—a very characteristic instance—the brain was not examined; Case V. recovered; in Case VI. the brain was not examined; in Case VIII., also a marked case, in which there were flushing of the face, occasional spasmodic contractions of the facial muscles, contracted pupils, undulatory motions of the body, and tossing of the arms, an examination was refused; in Case IX., in which there were mental symptoms, facial paralysis, ptosis, and hemiplegia, the patient recovered; in Case XI. recovery took place, as it did also in Case XII., so that in only six were there post-mortem examinations of the encephalon.

Dr. Da Costa does not doubt the existence of rheumatic meningitis, but he contends, and I think successfully, that all cases of cerebral disorder, originating during the course of articular rheumatism, are not cases of meningitis, and that in some cases there are actually no abnormal post-mortem appearances.

[1] Art. "Méningite," in "Dictionnaire de Médecine," Paris, 1839.

[2] "Traité des maladies des enfants," Paris, 1853.

[3] "A Practical Treatise on the Diseases of Children," Philadelphia, 1870, p. 464.

[4] "Recherches sur l'inflammation de l'arachnoide," Paris, 1821.

to be exposed to strong and direct heat; among the latter are excessive intellectual exertion, and abuse of alcoholic liquors. Tertiary syphilis, gout, and rheumatism, are likewise predisponents.

Larrey[1] states that in the retreat of the French army from Russia, the soldiers, who had endured the most terrible sufferings from hunger and cold, were attacked, on their arrival in Königsberg, where they had ample food and warm quarters, with cerebral meningitis, which in general proved fatal. This result was probably due to the operation of many causes besides prolonged exposure to a low temperature, among which the sudden removal of the mental tension maintained by the exigencies of the situation in which the army was placed, was not the least.

Of exciting causes, injuries of the head from falls or blows of different kinds stand first. Next is exposure to the direct rays of the sun, or other source of great heat, and then recession of an exanthematous affection, such as scarlatina, measles, or erysipelas, and the irritation of dentition, or intestinal worms.

Acute cerebral meningitis sometimes prevails epidemically. Such was the case with the series of instances which came under Larrey's observation, and others have been noted.

Diagnosis.—Acute meningitis may be confounded with partial or circumscribed encephalitis, but the distinction is made by considering that in the latter the headache is less severe, the delirium less marked, and the convulsions and contractions weaker. Moreover, the febrile excitement is much greater in acute meningitis than in partial encephalitis, and the whole disease more pronounced.

The meningitis of the aged bears a considerable degree of resemblance to cerebral softening; but the fact that the first-named affection is more rapid in its progress, and is not preceded by symptoms due to other morbid conditions, will generally enable the practitioner to make a correct diagnosis.

From delirium tremens it may be distinguished by the history of the case, by the greater tendency to insomnia exhibited in alcoholism, and by the general character of the delirium. The febrile excitement of acute meningitis, the pain in the head, the heat of the skin, the absence of clammy perspiration, and the increased temperature, as shown by the thermometer, are conclusive diagnostic marks.

From typhoid fever meningitis is diagnosticated by the existence in the former of meteorism, abdominal tenderness, and petechiæ, by the facts that the headache and febrile excitement are less, and that diarrhœa is present and vomiting is not.

Prognosis.—This is always grave. Occasionally death takes place in a very few hours, and generally before the tenth day. When the disease is prolonged beyond this latter period, the prognosis become more favorable. The occurrence of strabismus or other paralytic affection, lessens the hope of a favorable termination. Prof. Flint, however, has cited two cases occurring in the hospital practice of himself and Dr. Thomas, in which there were strabismus, hemiplegia, and coma, both of which recovered. He also cites another case in which there was strabismus, and in which recovery took place. Hiccough is an unfavorable event.

---

[1] "Mémoires de chirurgie militaire et campagnes," Paris, 1817, tome iv., p. 189.

Of the thirteen cases observed by myself, eleven died. In all of these fatal cases there was strabismus. In the two cases which recovered there was no squinting. The deaths in the fatal cases all occurred before the tenth day, and two took place before the end of the third day.

Morbid Anatomy.—If death occurs during the second stage of the disease, the most marked appearance found in the membranes is redness from increased hyperæmia. If, however, it is delayed till the third stage, thickening and opacity of the membranes and adhesions to each other, and of the pia mater to the brain, and effusion of serum, are the prominent features. In a case in which I made a post-mortem examination in the summer of 1870, and which was caused by the great heat of the season, there was an extensive collection of bloody serum in the cavity of the arachnoid, and the pia mater was so adherent as to bring with it a layer of the gray matter of the brain as it was stripped off.

The fluid may consist solely of pus, or this may be mingled with serum in all proportions. The pus, with the fibrine of the exuded serum, often forms thin plates of membraniform texture, which are scattered over the surface of the inflamed region or may entirely cover it, and which are of the nature of false membranes.

If death has taken place late in the course of the disease, evidences of the implication of the cerebral substance will generally be discerned. These consist in the gray matter becoming of a pinkish color, and the white, when cut, showing numerous puncta vasculosa. The ventricles rarely contain any considerable amount of fluid, and are often entirely empty. The latter was the case in the instance above mentioned.

Pathology.—The symptoms of the first and second stages are due to congestion; those of the third mainly to effusion and consequent pressure.

An important question connected with the pathology relates to the determination, from the symptoms, what part of the brain is the seat of the lesion. The upper convex portion of the hemispheres is intimately related to the purely intellectual functions of the brain, while the under surface, or base, is connected with the motility of various parts of the body. Thus, if the inflammation be strictly limited to the upper surface of the brain, the predominant symptoms are those involving intellectuality, and consequently there is delirium, marked by incoherence of ideas and irrationality of language. If, on the contrary, the base of the brain alone is affected, the chief manifestations of disease are seen in the muscular system, and there are contractions, spasms, convulsions, and paralysis. When the morbid action extends to both regions, there is a combination of these phenomena.

But, as Jaccoud[1] states, there are some stubborn facts which stand in the way of the unreserved acceptance of the law laid down, for it occasionally happens that the symptoms are not in direct relation with the seat of the lesion. Thus, in the case the post-mortem examination of which I have referred to, there had been spasms and paralysis, yet the convex surface of the right hemisphere was alone involved, and that to an extent not exceeding a third the size of the hand. Jaccoud explains such cases by attributing to the cerebral symptoms a double origin; one set being due directly to the part affected, the other resulting from secondary reflex excitation.

But a better explanation is to be found in the experiments of Fritsch and Hitzig,[2] and of Ferrier,[3] by which it is shown that there are distinct centres of muscular motion situated in the cortical substance of the brain, and that hence when this is irritated, as in the case referred to, by the occurrence of inflammation, spasms will be initiated in those muscles which are in direct relation with the centre implicated. It therefore is not necessary to frame an hypothesis to account for occurrences which are readily explained by facts.

Another fact should also be taken into consideration. In acute cerebral meningitis there is very frequently a large effusion of serum or an extensive formation of pus. If either be collected on the upper convex surface of either hemisphere, the pressure exerted through the intervening brain-substance upon the motor tract at the base must produce more or less derangement of motility on the opposite side of the body.

Guyot,[4] who has given very careful study to the localization of the lesion from a consideration of the symptoms, declares that it is possible to define the seat very accurately, but his manner of looking at the subject places it in altogether a different position from that which Jaccoud gives it, and which is not inconsistent with the investigations of Fritsch and Hitzig, and of Ferrier. Thus, tracing the fibres of the motor tract through the white substance to the convex surface of the hemispheres, he associates lesion of this region, not only with disturbances of ideation, but with derangement of motor functions. In this view he is supported by the experience of MM. Parent-Duchatelet and Martinet,[5] who state that in eight subjects who had exhibited hemiplegia, or the beginning of paralysis on one side of the body, they had discovered, on post-mortem examination, effusion on the convexity of the opposite hemisphere.

---

[1] *Op. cit.*, p. 212.

[2] "Ueber die electrische Erregbarkeit des Gehirns." *Archiv für Anatomie und Physiologie*, von Du Bois-Reymond und Reichert, 1870, p. 300, *et seq.*

[3] "Experimental Researches in Cerebral Physiology and Pathology," "West Riding Lunatic Asylum Medical Reports," vol. iii., 1873, p. 30.

[4] "Du rapport des symptoms avec les lesions dans la méningite." Thèse de Paris, 1859.

[5] *Op. cit.*

When, however, the lesion is limited to the base, the functions of the hemispheres will not be affected, except upon the principle of reflex irritation, or of the transmission of pressure. It is evident, however, that further researches, founded upon post-mortem examinations, are necessary to the satisfactory solution of the interesting questions involved.

Treatment.—To afford any chance of a favorable result, the treatment should be energetic from the first.

General bloodletting may be practised with advantage in subjects of good constitution and of the middle period of life. As many as twelve or sixteen ounces may be taken from the arm if the pulse is hard, the cephalalgia intense, or the delirium furious. Leeches applied behind the ears or to the inside of the nostrils are more generally of advantage. The same may be said of cups to the nucha.

The hair should be cut off short, and ice kept constantly applied to the scalp during the first and second stages. It is better than the cold douche, for the reason that it is almost impossible to continue the latter without intermissions, during which the head again becomes hot. Compresses wrung out of cold water will not answer; they soon get heated, and act as poultices. Irrigation, by a small stream of ice-water falling from a vessel placed above the head of the patient, is a useful means of applying cold, but is often inconvenient.

The experiments of Dr. Benham[1] appear to show that cold applied to the head has no material effect in reducing the intra-cranial temperature, or in lessening the amount of blood flowing to the brain. But it must be borne in mind that, though cold applied to the scalp may not reduce the normal intra-cranial temperature, it may exercise a very different influence over temperature which is abnormally high, and that his experiments with Ludwig's Strohm-uhr were but three in number, that the cold was only applied for thirty minutes, and that it is quite doubtful if the Strohm-uhr affords the best means, under the circumstances, for determining the quantity of blood flowing to the brain. In actual experience, we find that the sedative influence of cold to the head is as well-established a fact as any other in therapeutics, and, though it may fail, as every other remedy does some time or other, to produce its expected effect, that fact should be no reason against our employment of it in cases in which it appears to be indicated. In acute cerebral meningitis, I have repeatedly seen the violence of the symptoms mitigated by the agent in question, but, in order to obtain this result, it should be kept persistently applied in the forms above mentioned.

---

[1] "On the Therapeutic Value of Cold to the Head," "West Riding Lunatic Asylum Medical Reports," vol. iv., 1874, p. 152.

Purgatives are generally advantageous and should be effective. Nothing is better than croton-oil, although calomel and podophyllin, grs. x with grs. ij, make a good combination for the purpose.

My experience has satisfied me of the good effects of mercurialization. I have administered calomel in doses of a grain every two hours until the breath became fetid, and I am sure the effect has been beneficial.

The iodide of potassium is well spoken of by Dr. Flint,[1] who says he has witnessed the good effects of the drug in several cases. Dr. F. R. Lyman[2] has reported two cases in which it formed a prominent feature of the treatment, and in which recovery took place.

Within late years in the few cases of acute cerebral meningitis that have been under my charge, I have found the greatest benefit from the bromide of potassium, and the three cases that recovered were instances in which it was administered in large doses. The theory upon which its employment is based has already been fully considered in the chapter on cerebral congestion. It should be administered in doses of at least thirty grains three or four times a day, from the very beginning of the affection to the end of the second stage or the appearance of coma, should this symptom supervene.

The head should be kept well elevated, the chamber cool, and well ventilated, the light in a great measure excluded, and the utmost quiet enjoined.

The food, without being stimulating, should be nutritious. Nothing is superior to strong beef-tea, made either from fresh beef or from some one of the extracts in the market.

In the third stage the treatment should be almost the reverse of that indicated as proper for the first and second stages. The mercury, iodide of potassium, bromide of potassium, ice to the head, and purgatives should be omitted, and attention should be given to the maintenance of the strength. To this end brandy, whiskey, or other alcoholic liquor, should be administered in such quantities as the occasion seems to require. It often happens in this stage that the delirium and excessive motility return. It must be remembered that this is not from any renewal of morbid processes within the cranium, but is entirely due to debility. At the moment of writing this, a young lady of this city is under my charge for acute cerebral meningitis, whom I did not see till the third stage was well advanced, and who for several days previously had exhibited a return of the delirium, for which depletive measures and hydrate of chloral had been employed. The free administration of brandy, champagne, and beef-tea, soon dissipated the symptoms of relapse, and she bids fair to recover.

---

[1] *Op. cit.*, p. 601.

[2] *American Medical Times*, 1862, p. 334.

Blisters may be used in this stage with advantage. They are best applied between the shoulders, and should be six or eight inches square.

In the rheumatic form of the disease little special treatment is necessary. It is, perhaps, advisable to endeavor, by means of blisters or other revulsives, to bring back the disease to the joints.

In the acute meningitis of the aged, active depletive treatment is not so generally admissible, and if apparently indicated should be carried out cautiously. It may even be proper to treat some cases with stimulants from the very first.

CHAPTER IX.

CHRONIC CEREBRAL MENINGITIS.

Although it is scarcely possible, for reasons given in the preceding chapter, to determine from the symptoms the exact seat of the morbid process in an attack of acute cerebral meningitis, we are often able, in the chronic form of the disease, to make the differential diagnosis with sufficient accuracy. I shall therefore consider the affection according to its location under the heads of Chronic Verticalar Meningitis, and Chronic Basilar Meningitis, the terms being applied respectively to chronic inflammation of the membranes of the superior surface or vertex of the brain, and chronic inflammation of the membranes of the inferior surface or base of the brain.

I.—CHRONIC VERTICALAR MENINGITIS.

This disease may be the consequence of an attack of acute cerebral meningitis, or may originate without being thus preceded. The latter is the usual mode of development.

Symptoms.—The symptoms of chronic verticalar meningitis are in some respects similar to those of general paralysis, an affection which will be fully described as one of the forms of insanity; and they also resemble those evolved during the course of softening, limited to the convex portion of the brain.

Among the physical symptoms headache occupies a prominent position and is usually the first evidence of cerebral disease which attracts the attention of the patient. The pain is generally felt in the forehead, in one or both eyes, or at the vertex, and is aggravated by mental exertion, by the mere act of reading or fixing the attention, by muscular effort, or by a dependent position of the head. It is not usually very intense, but is characterized by persistency. There are frequent attacks of vertigo. Somnolency is generally present, and there are trembling, defective articulation, weakness of the limbs, spasms of particular muscles or groups of muscles, paralysis of the bladder or of the sphincters of the bladder and rectum, producing involuntary discharges of urine and fæces, weakness of the memory, especially as regards words, and a general enfeeblement of the mental faculties. Occasionally there are epileptic convulsions.

Paralysis of the whole of one side of the body may ensue, or the loss of power may be confined to a single limb, or to a group of muscles. Anæsthesia may be present, either general or local, or there may be neuralgic pains in various parts of the body, sometimes of a very persistent character. The ocular muscles are not often implicated, either by spasm or paralysis; and the special senses, except that of general sensibility, are not usually impaired. Convulsions of an epileptiform character are not uncommon.

Unless the cortical substance of the brain participates in the morbid action there is not ordinarily marked mental aberration, although there is a general failure of mental power. Under the name of "general paralysis,"[1] and subsequently of "chronic, diffused periencephalitis,[2]" Calmeil described a disease which is now well known, and in which the cortical portion of the upper part of the cerebrum is in a condition of chronic inflammation, the membranes of the region being also involved. But the peculiarities of general paralysis are so well marked as to necessitate separate description.

The ophthalmoscope does not, in this affection, generally reveal any very notable changes in the fundus of the eyes. Occasionally, where there is reason to suspect its existence, there is ischæmia papillæ, and still more rarely neuro-retinitis. As Dr. Allbutt[3] has remarked, the optic nerves in drunkards affected with meningitis of the convex surface of the brain "are often degenerated, and the vessels injected, but these effects do not seem to be due to any meningitic process." When, however, the meningitis is complicated with inflammation of the cortical substance of the brain, neuro-retinitis is a frequent accompaniment.

The general health participates more or less in the disturbance. The stomach is irritable, and vomiting is frequent, the bowels are usually obstinately constipated, and the urine is scanty and high-colored, often containing oxalate of lime and an excessive amount of uric acid.

As the disease advances, the mental and physical symptoms become more and more pronounced. The mind is weaker, delirium is not infrequent, convulsions occur oftener, and the paralysis extends and becomes more profound. Blindness from pressure upon the optic nerve may result. A state of continued coma now supervenes, during which the patient expires, or death takes place in convulsions.

---

[1] "De la paralysie considérée chez les aliénés," Paris, 1826.

[2] "Traité des maladies inflammatoires du cerveau," Paris, 1859.

[3] "On the Use of the Ophthalmoscope in Diseases of the Nervous System," etc., Lo don and New York, 1871, p. 108.

The duration of the disease varies from two or three months to one or more years.

An interesting case of meningitis affecting the membranes at the convexity of the brain, is that of the eminent Swiss *savant* De Saussure, related by Dr. Odier.[1]

For many years M. de Saussure had been accustomed to great bodily fatigue, and to various degrees of atmospheric pressure, encountered in the many ascents of mountains he had made. He had been subject to an aggravated form of dyspepsia, and to repeated large losses of blood from hæmorrhoids.

At the end of the year 1793, after having lost his fortune, and experienced a good deal of mental disturbance from the unsettled condition of the national affairs, he was suddenly seized with vertigo, which was followed by distinct sense of numbness in the left arm and cheek. The vertigo did not last long, but nothing could relieve the feeling of numbness or torpor. Blisters, purgatives, tonics, and anti-spasmodics, were employed in vain. The affection of the arm seemed to be seated entirely in the sentient nerves, for the patient retained his strength, could perform all kinds of movements, but could not distinguish easily what he was touching. It seemed to him as if sand were interposed between his fingers and the bodies with which he brought them in contact. The sensation experienced was rather painful than otherwise, so that he was indisposed to use his hands unless they were protected with gloves. A similar feeling existed in the cheek and mouth on the same side, which, on passing his hand over his face, formed, in the most unpleasant manner, a well-marked line of demarkation between the right and left side. In other respects he was well; his general health was not impaired, and he retained for a long time his presence of mind and the fullness of his intellectual powers. Many months were passed in this state, during which a great variety of remedies were tried, such as cold and warm bathing, electricity, arnica, valerian, blisters, embrocations, artificial and natural thermal waters, change of regimen, traveling, etc., but all in vain. The disease became worse and worse; always, however, by starts, the attacks being more or less violent and complete. One of the most violent was occasioned suddenly at Bourbon, by a shower-bath employed too warm. The attack produced by it was so complete that the whole of the left side, from the leg to the tongue, was affected. His articulation became by degrees indistinct and unintelligible. His legs, especially the left, became weaker, and his gait was staggering, and he found it almost impossible to maintain his equilibrium and to direct his steps as he wanted. He experienced peculiar difficulty in passing through doors, even when they were wide open, and no descent or ascent to make. As he approached a door he balanced himself, and quickened his motion as if he had to make a dangerous leap or a bad step to get over; when it was done he recovered his equilibrium, crossed the room, but had the same trouble in order to get to another apartment. Day by day the disease advanced; the intellectual faculties became perceptibly weaker; incontinence of urine supervened. The evening before his death he seemed to enjoy his supper, but was restless during the night; toward morning his head leaned to one side, he breathed with more difficulty than usual, and expired without agony.

---

[1] "An Account of the Illness and Death of H. B. de Saussure, late Professor of Philosophy at Geneva," *Edinburgh Medical and Surgical Journal*, vol. ii., 1806, p. 393.

On opening the body thirty-two hours after death, the dura mater was found adherent to the cranium, particularly along the longitudinal sinus, but that deviation from the natural condition was not considered of importance, it being often met with unassociated with intra-cranial disease. Between the pia mater and the arachnoid there was found a considerable effusion of a bluish gelatinous substance. In various places there were circular spots of a gray yellowish color about two or three lines in diameter. These seemed as though they penetrated into the membranes, though susceptible of being detached from them like small separate spheres surrounded by a little circular margin of a dark-red color. At first sight these spots were taken for hydatids, but closer examination showed that the red margin was a blood-vessel connected with other vessels, and convoluted in the form of circles. There were no separate pouches or solutions of continuity in the membranes, only they were more transparent in those places than in others. The serosity underneath communicated freely with that which was diffused over all the surface of the brain, both having the same color and qualities. On opening the membranes the serous effusion ran off like water. The effusion existed not only over the surface of the cerebrum, but also over that of the cerebellum. The ventricles also were distended with a similar fluid. The examination of the brain presented nothing more of importance except that it was flattened on the surface and deeply furrowed by arteries. The total duration of the disease was five years, although the beginning may have been anterior to the apparent time of origination, as it was stated that Prof. de Saussure, long before his death, had often mistaken one word for another in conversation, and was so unconscious of his error as to get angry when not understood.

Dr. Odier attributed the death of the patient to the effusion of a large quantity of serum into the ventricles and between the membranes of the brain. That this effusion resulted from chronic meningitis is scarcely a matter of doubt.

Gintrac[1] cites the following case: "A young man sixteen years old, very tall, was attacked in December with feebleness of sight, strabismus, dilatation of the pupils, diplopia, and headache ; pulse natural, constipation, epistaxis; convulsions, with foaming at the mouth; coma and stertor, which were relieved by bleeding from the temporal artery, but which returned twenty-four hours later. Delirium supervened, characterized by violent language, and attempts to strike and bite those around him ; pulse frequent. The wound in the artery being reopened, repeated losses of blood occurred, and the convulsions returned. Sight weakened, ideas confused, appetite voracious, general debility, but power of walking, of comprehension, and of speech, remained. Then somnolency, attended with spasmodic movements of the muscles, especially of those of the face, appeared. The face was red and swollen, especially on the left side. Death occurred in violent convulsions two months after the beginning of the disease.

---

[1] *Op. cit.*, tome ii., p. 626. Quoted from Bruce, "Medico-Chirurgical Transactions," London, 1818, vol. ix., p. 280.

"The cerebral blood-vessels were found to be very much injected. On the left anterior lobe there was a slight effusion of blood; a little serum in the ventricles; substance of the brain firm; numerous purulent spots along the line of superior longitudinal sinus."

M. Casimir Broussais[1] submitted to the Académie de Médecine a pathological specimen with the history, of which I give the main points:

Lozeray, a *sapeur pompier*, twenty-two years old, entered the hospital Val-de-Grace August 1, 1840. Six days previously he had been attacked with headache and slight fever. The evening of his entrance he was bled. He improved, the pain disappeared, and his appetite returned. On the 7th of August he had a relapse; hardly answered the questions addressed to him; remained motionless in bed; was entirely paralyzed in the right arm and leg; was again bled. The next day, being comatose, venesection was again practised, and twenty leeches were applied to the temples. On the 9th the paralysis had disappeared, but, as he was still comatose, another venesection was performed, and fifteen leeches were applied to the neck over the jugular vein. On the 10th was bled again; still comatose, and the right arm contracted. On the 12th had epileptic paroxysms, during which it was remarked that one side was more convulsed than the other; coma profound; eighteen leeches to the jugulars; 14th, 15th, and 16th, same symptoms; an enormous bed-sore on the sacrum. On the 18th coma less complete; epileptic convulsions, especially in the night. From this time he continued to improve till the 28th, when coma again supervened, and on the 29th he died.

On post-mortem examination the dura mater was found healthy. On being incised, a quantity of sero-purulent fluid escaped. The membrane was adherent to the brain, principally on the convex surface, and especially on the right side, so that it was impossible to detach it entirely without rupture. On the right side it formed a sac extending over about three-fourths of the convex surface, containing from two hundred to two hundred and fifty grammes of a greenish-white sero-purulent fluid. Another sac, containing from fifty to sixty grammes of this fluid, existed on the left side.

---

[1] "Bulletin de l'académie royale de médecine," tome v., 1840, p. 564.

The dura mater was removed, and it was ascertained that this fluid came from the cavity of the arachnoid and from the meshes of the pia mater.

In the case of a gentleman under my charge there was intense headache as the first prominent symptom, followed by epileptiform convulsions, and varying degrees of paralysis, both of motion and of sensation on one side of the body and again on the other. When I first saw him the optic nerves had been so injured by the pressure from effused fluid as to cause complete blindness. Light could not be distinguished from darkness. The ophthalmoscope showed extreme atrophy of both nerves, probably either the result of pressure or the consequence of neuritis from extension of the cerebral disease. The accumulation of fluid was so great as to force open the bi-parietal, the fronto-parietal, and the occipito-parietal sutures. Under treatment the excess of fluid disappeared, the pain ceased, and he acquired the power of vision to such an extent as to enable him to tell light from darkness, and even to make out the figures on a bright carpet. He died, however, about six months after leaving New York, of cancer of the stomach. There was no post-mortem examination of the brain, or none that was reported to me, but I am strongly of the opinion that the disease was chronic meningitis of the convexity of the brain, resulting in a large effusion of serum.

Causes.—The etiology of chronic cerebral verticalar meningitis is often difficult to make out. Sometimes, however, the affection is the result of an acute attack. At times it clearly originates from blows or falls upon the head, and again it is caused by exposure to the heat of the sun or to artificial heat. There is certainly a form of chronic inflammation of the membranes of the convex surface of the brain, which is due to the extreme heat of the sun, not necessarily to the action of the direct rays, and which is characterized by the symptoms I have specified. I see some cases of this every year in New York, and have witnessed several similar instances in cooks and others whose occupations necessitated the exposure of the vertex to intense or long-continued heat.

The affection in question may also be induced by mental influence, especially anxiety and other forms of emotional disturbance; and this category of causes is probably the most influential of all others, with the single exception of excessive alcoholic potations. So far as our knowledge extends, this last is the most common factor in the causation of chronic verticalar meningitis.

Syphilis is another influential cause, though generally, as we shall see hereafter, it acts preferably upon the basilar portion of the membranes.

It is probably sometimes induced by rheumatism and gout, and certainly occasionally by tubercular deposit, but when arising from this last-named cause it is not to be confounded with tubercular cerebral meningitis, the seat of which is in the membranes at the base of the brain, and which is otherwise differently characterized.

Diagnosis.—This is often impossible to be made out, with even a moderate degree of exactness, and is always more or less difficult. The affection may be confounded with inflammation and softening of the cortical substance of the cerebrum, and the most careful study will in many cases fail in discriminating between them. The difficulty is frequently heightened by the fact that the two diseases coexist. But we are much assisted by a thorough investigation, not only of the symptoms, but of the causes. For instance, a category of phenomena such as has been given, resulting from exposure to intense heat, is generally due to chronic inflammation of the membranes of the superior surface of the brain, and the same may be said of syphilis. When, however, the symptoms follow undue mental exertion or emotional excitement, the distinction is more difficult, and indeed in such cases the substance of the cortex is usually also involved.

In general, the pain which is so prominent a feature in inflammation of the membranes, is not so marked an accompaniment of softening, while in the latter the mental disturbance is greater than when the morbid process is confined to the meninges. From inflammation of the membranes at the base of the brain, the affection under consideration is distinguished by the almost constant absence of ocular paralysis, and by the fact that the seat of the pain is different, and that the mind is more decidedly involved.

The ophthalmoscopic appearances will suffice for the diagnosis from anæmia or hyperæmia of the brain, or from megrim or neuralgia, even if the other points in the clinical history are not sufficient.

Prognosis.—The prognosis in cases of chronic inflammation of the meninges of the convex surface of the brain is decidedly unfavorable, unless a syphilitic origin can be made out, in which event the prospect of recovery is good. But even in such a case the disease must be early subjected to proper treatment, for the disposition to extend to the substance of the brain which the affection so often manifests, and the fact that new formations are liable to be produced and to exert an abnormal influence upon the nerve-tissue, very greatly increase the probability of an unfavorable result.

Nevertheless, I am satisfied that even where there is no suspicion of syphilis, chronic verticalar meningitis is sometimes successfully combated. This point will be further considered under the head of treatment. In the mean time I quote the following case from Dr. E. L. Fox,[1] of Bristol, England, in which a post-mortem examination gave evidence of the previous existence of the disease in question. It is possible there was a syphilitic taint in this case, though nothing is said on the subject:

"The patient, a young man, had died of an attack of hæmorrhage, from rupture of the right middle meningeal artery, but the dura mater, all over the convex surface of the hemispheres, was somewhat adherent to the subjacent arachnoid, while the arachnoid was thickened and yellow all over. This patient had been under Mr. Parker's care a year before, with great pain all over the upper part of the head, without any delirium, and had been treated, with entire success, with iodide of potassium. In this case, therefore, arachnitis had existed without any lesion of the cerebral matter itself, and without delirium."

Morbid Anatomy and Pathology.—The essential features in the morbid anatomy of chronic cerebral verticalar meningitis are hyperæmia of the vessels and a new formation of connective tissue by which the membranes adhere to each other and to the brain, and by which they are rendered opaque, and thicker than normal.

In addition, there may be deposits of exudation on the convexity of the brain which, though intimately connected with the alterations of the membranes, are yet distinct from them. These, as characterized by Gintrac,[2] may consist of serum effused under the arachnoid, of a thick, gelatiniform, discolored fluid in the same situation, of pus contained either in the cavity of the arachnoid or infiltrated into the meshes of the pia mater, of false membranes formed in the cavity of the arachnoid, non-adherent, adherent to one or other layer of this membrane, or double, composed of an external layer of the arachnoid and an internal, adherent to the visceral lamina, thus constituting cysts which may contain blood-serum or other matter.

Of one hundred and sixty-seven cases of meningitis of the convexity of the brain collected by Gintrac—in which, however, the distinction between the acute and chronic forms of the disease is not drawn—the relative proportion of morbid conditions was as follows:

Injection, opacity, or thickening of the membranes	9
Serous exudation	33
Gelatiniform exudation	14
Pus	30
False membranes	81
Total	167

Fox[3] has very clearly shown that tubercle may be associated with chronic meningitis of the convexity of the brain. The following case, which I cite from him, is so interesting in several respects, that I quote it in full, so far as the description relates to the brain:

---

[1] "Clinical Observations on Acute Tubercle," "St. George's Hospital Reports," London, 1869, vol. iv., p. 61.

[2] *Op. cit.*, tome ii., p. 604.

[3] *Op. et loc. cit.*

"CASE XXII.—Henry B., aged twenty-four, tailor; ill one month with pain in the forehead; no cough. When first examined in recumbent position, a sharp, blowing, systolic murmur was heard at the base of the heart, traveling up toward the left shoulder; a little later he had sickness, then intense pain, chiefly at back of head. Head jerks backward at every beat of the heart; much cerebral throbbing. Temporary relief from blisters, cold to the head, and purgatives; but eventually more sickness, diplopia, which, however, was intermittent, and increased headache. Then almost total freedom from pain, and all morbid symptoms, and he was able to be out; but he died suddenly in a fit, three months from the commencement of his illness. No bronzing of skin.

"*Post-mortem Examination.*—Dura mater externally seemed healthy; internally it was firmly adherent to the subjacent tissues at the spots below mentioned; veins of convex surface of hemispheres tinged with blood. On left hemisphere, about middle of brain, was a spot of tuberculous matter the size of a filbert, which seemed to be immediately connected with the vessels of the pia mater, to have become adherent on the one side to the dura mater, and on the other to have extended through the gray matter for a few lines into the white. The two lateral and third ventricles much distended with clear fluid, containing a few small, white flakes. Foramen of Monro enlarged sufficiently to contain a small nut. Walls of ventricles very soft; optic thalami tolerably firm. Corpora striata excessively pulpy; pons and medulla oblongata everywhere rather soft. On anterior lobe of right hemisphere, just on the lateral surface, was another tuberculous spot the size of a nut. On the external surface of the cerebellum, close to the flocculus on left side, though not involving it, was a large mass of tubercule, dipping into the structure of the cerebellum, and uniting this organ to the posterior lobe of the left cerebral hemisphere. More than three-quarters of the left half of the cerebellum were occupied by large vessels of the same growth, which apparently had grown separately, and by gradual increase of size had at length become one mass. The dura mater was adherent over a great part of this side of the cerebellum, and the cerebellar structure that remained was almost diffluent. The other side of the cerebellum was also much softened."

This case is remarkable, not only for the intermittence in the symptoms, to which Dr. Fox calls attention, but also for the lightness of the phenomena when compared with the severity and extent of the lesions. Such remissions in the manifestations of cerebral disease as were exhibited in this case, though not unusual, are, in the present state of our knowledge, not easy of explanation. For it is very evident that there was a steady advance of the morbid processes up to the very instant of death, and yet the patient died suddenly, having up to that time passed through a period of almost entire freedom from pain and all morbid symptoms.

I am tempted also to cite the next case from Dr. Fox's memoir, on account of a like slightness of symptoms existing in connection with extensive cerebral lesions.

CASE XXIII.—Catharine S., aged thirty-one, servant; single; pale, lean woman; has had vertigo and pain in back of the head for five weeks; no sickness, no rigors, pulse now very feeble and hurried. Tongue coated; skin hot; no sickness until eight days after admission, and she coughed first on the ninth day. Became delirious, but was always capable of answering questions reasonably, and the chief symptom was a gradually increasing weakness of pulse. Sank quietly out of life, without coma, on the twenty-second day after admission, having had no convulsions throughout, and no cerebral respiration until the last day of life.

"*Post-mortem Examination.*—Cranium: Arachnoid, and subjacent tissues on convex surface of the hemispheres, contained much clear fluid, but were otherwise natural. Between the cerebral hemispheres and the longitudinal fissure were a number of small, miliary tubercles, and at the lower part of this fissure the opposed hemispheres were adherent to each other by means of a mass of tuberculous matter the size of a nut. A small portion of similar matter was found at the upper part of the cerebellum, connected with the arachnoid. The venous tissue around these tuberculous masses was very much softened and ecchymosed. Two similar masses were also found in inner wall of posterior horn of each lateral ventricle. Ventricles full of turbid fluid, and their walls softened."

It sometimes happens that chronic inflammation of the membranes of the vertex of the brain exists without the occurrence of notable symptoms. Several such cases have come under my own observation in which, after death, the membranes were found thickened, opaque, and adherent, and in which, during life, no complaint of cerebral disturbance had been made. It is probable, however, that symptoms of such disturbance have existed, but have not been mentioned by the patient.

Treatment.—The treatment depends to some extent upon the cause, although the general management of the disease is not subject to any very essential variation, however it may originate. Thus the iodide of potassium is in all cases the agent most to be relied upon. When the affection is due to syphilis, or has followed syphilitic infection, the iodide must be administered with much more persistency and in larger doses than when not so associated. In all cases, however, it must be given in what may be called large doses, and must be continued for several months. In uncomplicated cases the quantity administered may be at first ten grains three times a day, gradually increased to thirty grains for each dose; but in syphilitic cases the doses will often have to be carried to eighty or even a hundred grains thrice daily. The iodide of potassium should, in my opinion, always be given in gradually-increasing doses. This is best effected by using a saturated solution of the medicine in water, each minim of which contains about a grain of the salt. For the first day ten minims may be given three times, for the second day eleven, and so on till the maximum dose, which it may be deemed proper to administer, is reached. I have several times had cases under my charge in which no sign of amelioration occurred till doses of from eighty to one hundred grains thrice daily were used.

Some one of the bromides may be very advantageously given in addition to the iodide of potassium. The bromide of calcium is to be preferred in almost all cases. It acts more rapidly than the others, and, notwithstanding the recent opinion of a German physician, more effectually. The doses should be about fifteen grains daily, and each dose may be given with that of the iodide of potassium. It must not be forgotten that these medicines must, when taken, be administered in a large quantity of water (half a tumbler, for instance). They act better, and are less liable to irritate the stomach, when they are well diluted.

Under the combined action of the bromide and iodide, the relief from all symptoms of intra-cranial disease is often very striking. This is especially apt to be the case when syphilis is at the bottom of the morbid process.

Relative to the propriety of administering mercury in chronic cerebral verticalar meningitis, much depends upon the nature and duration of the disease. In non-syphilitic cases it is not indicated, nor in those instances in which the syphilitic infection is remote, but, where the primary disease is recent, mercury is of service as an addition to the other measures. It may be given in the form of the biniodide, or the bichloride, in doses of the sixteenth of a grain two or three times a day.

For the relief of the pain, which is sometimes very severe, a pill containing half a grain of codeia may be prescribed with advantage, as often as required.

In regard to local medication, I am inclined, from more recent experiences, to believe that blisters applied to the nape of the neck are occasionally beneficial. As a rule, however, I do not employ them, or any other revulsive or counter-irritant means.

The patient should be instructed not to over-exert the mind, to avoid all causes of excitement, mental or physical, and live in strict accordance with hygienic principles.

CHRONIC BASILAR MENINGITIS.

Chronic basilar meningitis is very seldom the consequence of an acute attack, probably mainly for the reason that acute inflammation of the membranes at the base of the brain is almost invariably a fatal affection.

Symptoms.—Although there is generally pain from the very inception of chronic basilar meningitis, the first very decided symptom is sometimes an epileptiform paroxysm. Or there may be convulsive movements of a limb, a group of muscles, or a single muscle, unattended with loss of consciousness.

Again, there may be tonic spasms of the muscles of one or more of the extremities, especially of the arms; or the muscles of the neck may be similarly affected, causing the head to be fixed in an abnormal position. The individual muscles of the face are not usually involved.

But ordinarily the primary serious indication of intra-cranial disease is paralysis. This may appear in the head, arm, the hand, or a single finger; or one side of the tongue may be affected, giving rise to defective articulation, and to a deviation toward the paralyzed side when the tongue is protruded, or the muscles supplied by the seventh nerve may be affected and facial paralysis be produced. In the great majority of cases, however, some one of the motor nerves of the eyeball is first involved in the morbid process, and this is generally the third nerve of one side, resulting in ptosis, external strabismus, and diplopia, dilatation of the pupil, and defective power of accommodation.

Sometimes the implication of the third nerve is not complete. Thus, there may be paralysis of the levator palpebræ superioris muscle, producing ptosis, or the internal rectus muscle of the eyeball may be paralyzed, causing the globe to be rotated outward by the uncompensated action of the external rectus, and as a consequence producing double vision; or, what is more rarely the case, the superior or inferior rectus, or the inferior oblique, may lose the power to act. In a few cases, the only indication of the affection of the third nerve is dilatation of the pupil.

The fourth nerve may be paralyzed, and then the loss of power is limited to the superior oblique muscle, and the ability to rotate the eyeball outward and downward is impaired; and again, the lesion is only manifested as regards the sixth nerve and the external rectus muscle, so that internal strabismus is the result. Occasionally the first sign of the disease is aphasia, with or without vertigo, confusion of ideas, or loss of consciousness.

It not infrequently happens that pain of a very severe character is for a long time the only symptom which disturbs the patient. It may be located in some part of the head, or may be referred by the patient to the face, and is often regarded and treated as ordinary neuralgia. The chief features of this pain are its intensity and persistency. I have known it to last, without interruption, night and day, for over four months, driving its subject to the verge of insanity, and causing him to entertain serious thoughts of suicide.

In a few of the cases which have come under my observation, the principal symptom was anæsthesia of certain portions of the cutaneous surface. The skin of the face appears to be particularly liable to this phenomenon, although I have seen it extend throughout the whole of one side of the body; again, confined to the lower extremities; and at other times to the trunk, or upper extremities. In one case this was unaccompanied by paralysis of motion anywhere, but in the others the muscles, or some of them supplied by the third nerve, were paralyzed. In a case reported by Pétrequin,[1] and cited by Lagneau,[2] of syphilitic necrosis of the frontal bone, and in which there was certainly also chronic basilar meningitis, the lower limbs were deprived of sensibility for two months.

Vertigo is almost always a prominent symptom, and may be so intense and persistent as to prevent the patient walking without support. At times it is impossible for the recumbent position to be abandoned, even for an instant, without the supervention of severe dizziness; at others it occurs unexpectedly, and may be the cause of the individual falling.

The eyesight is often impaired from a very early period. This may be due to paralysis of the accommodation, resulting from loss of power in the iris and ciliary muscle, especially the latter; for, though the iris probably has some influence in effecting the adjustment of the lens for different distances, it is in the ciliary muscle, as Von Graefe has shown, that the function mainly resides. The defect in question is shown by the difficulty which the patient experiences in distinguishing near objects. There is no trouble in seeing images at a distance, but the effort to read, for instance, is unsuccessful—the lines of print appearing blurred—and always increases the pain in the head, besides inducing temporary pain in the eye. The exact degree of impairment of accommodative power may be ascertained by the use of Snellen's test-type, or still better by Galezowski's typographical scales.[3]

Or the asthenopia may be the result of the paralysis of the internal rectus muscle.

Again, the defective vision may be caused by the disturbance in the special nervous apparatus of the eye. Examination with the ophthalmoscope almost invariably reveals the existence of hyperæmia of the optic nerve and retina, and not infrequently of optic neuritis, caused by extension of the morbid process from the cerebral membranes to the optic nerve. Sometimes, as in cases to be cited presently, vision may be entirely lost from this cause; but, again, it is indubitable, as Dr. Hughlings Jackson has very definitely shown,[4] that a great degree of optic neuritis may exist, and yet the patient be capable of minute vision.

---

[1] *Gazette Médicale de Paris*, 1836, tome iv., p. 643.

[2] "Maladies syphilitiques du système nerveux," Paris, 1860, p. 413.

[3] "Échelles typographiques et chromotiques pour l'examen de l'acuïté visuelle," Paris, 1874.

[4] Among other places, in the West Riding Lunatic Asylum Reports, in a paper enti-

The sense of hearing may also become impaired or lost by extension of the inflammation so as to involve the auditory nerve. Several cases of the kind have come under my observation; and in one, which will be more specifically referred to hereafter, the function was very suddenly regained under appropriate treatment.

Although mental exertion of all kinds adds to the severity of the symptoms, it is not usually the case that the mind is primarily affected to any considerable extent. There may be periods of depression but these are generally the result of the physical phenomena—the pain, vertigo, paralysis, etc., the sensations arising from or the contemplation of which are calculated to disturb the mental equanimity. When, however, the mind is brought to bear upon any subject, the intellectual processes are as correct as ever, the only difference being that they cannot be long continued without the supervention of fatigue and an aggravation of the symptoms.

It quite often happens that the seat of chronic basilar meningitis changes, and with the transference there is an alteration in the locality of the symptoms. This is especially seen in the matter of paralysis. Thus, in the beginning, the third nerve may be paralyzed, and eventually the extension of the lesion leads to the implication of the fourth, fifth, and sixth. Cases in illustration of this point, which have occurred in my own experience, will presently be adduced. In the mean time, the following example from Sir Charles Bell[1] will prove of interest. The fact that Sir Charles mistook the real nature of the disease will not detract from its importance. It is reported as a "Case of Disease of the Nerves within the Orbit.

"Martha Symmonds, aged forty-one, Northumberland Ward. This woman was admitted into the hospital for a disease apparently seated in the left orbit. Nine months ago she had a paralytic stroke, attended with the loss of power in her left arm, neck, and face, on the same side. She lost also her power of speech, excepting only to 'babble,' as she says. She recovered from this attack, and went into service. About eight or ten weeks ago, she was alarmed by a commencing dimness in both her eyes, and she was obliged to leave her place on account of this dimness of her sight. Both her eyes were equally affected, and there was no redness or opacity perceptible in either of them. She placed herself under a medical gentleman, because she dreaded a return of the palsy. About six weeks ago, the upper eyelid of the left eye fell, and

tled "A Case of Recovery from Double Optic Neuritis." The case was probably one of chronic basilar meningitis, of syphilitic origin.

[1] "The Nervous System of the Human Body. Embracing the Papers delivered to the Royal Society on the Subject of the Nerves," London, 1830. Appendix, p. cv. Edition o 1844, p. 343.

she could not raise it. At that time she suffered great pain above the left eye, and the pain extended upon the left side of her forehead. She at the same time lost the vision of this eye, although she could distinguish by it the light of day from darkness. She could direct the motions of this eyeball as well as of the other at that time, and the appearance of the eye was natural.

"Five days before she was admitted to the hospital she experienced a violent, deep, throbbing pain in her left eye, and from that time the eyeball, as she says, became enlarged, until it projected considerably beyond the orbit. Two days before her admittance, she was totally blind in that eye, and was deprived of sensation on the surface of the whole eye, eyelids, the internal corner of the nose, and upon the left side of her forehead.

"At present her left eye is covered with its upper eyelid, and projects greatly from its natural situation. The lower eyelid is everted as a consequence of the projection of the ball of the eye, and the conjunctiva is tumid and projecting. She cannot raise the upper eyelid, although when it is raised with her finger she can squeeze it down again, and winks with a motion which corresponds naturally with that of the other eye. It may be a question whether the globe of the eye is enlarged, or only protruded. The pupil is unnaturally large, and the iris is without motion. She cannot move the eyeball in any direction. The whole eye is insensible; she has just had her lower eyelid scarified, and she was not sensible of pain. She allows us also to press with our finger on the surface of the eye, without complaining of any pain, or winking; although, as we said above, she can still wink, and does wink with this eyelid when the other eye is threatened.

"*October 6th.*—To-day some further examination was made of this woman's face and head, in order to ascertain the extent of insensibility. It was stated in our last report that she has lost sensation in the surface of the left eye and eyelids, in the corner of the nose, and upon the forehead. In these parts, she says that now the loss of sensation is less complete, because when she had her eyelid scarified, the other day, she felt pain, which she did not when it was scarified before. The eye also seems diminished in size.

"Besides those parts which we have already described as being affected, she has, in a partial degree, lost sensibility to touch in that part of her cheek which is just under the orbit, and downward upon the side of her nose, and upon the left side of her upper lip, and also within the cavity of the nose on the left side. However, when the point of the pin was brought near to the ear, or upon the skin which is over the lower jaw, she then was sensible of pain. A piece of linen was twisted so that it might be introduced into the left nostril; she allowed us to push it upward as far as we could, and, during this operation, she only remarked that she was sensible of its presence. Turning it about within her nostril did not make her sneeze. When we tried the same experiment on the other nostril, she was unable to bear the tickling produced by the loose threads of the cloth, before it was introduced into the nostril. Now she informed us that she is in the habit of taking snuff; and she is not only insensible to its usually agreeable effects, but unconscious of its presence in the left side of the nose. We next made her close her right nostril, and inhale strong spirit of ammonia; and then repeated the same experiment on the other nostril. There was a very obvious difference in the effects produced by the ammonia on the two sides of the nose. She told us she could smell the ammonia on both sides, but still she could not bear to hold the bottle containing the ammonia so long at the right nostril as we observed that she could at her left. When the bottle was placed under the right nostril, its pungency affected her almost immediately, so much that she could not bear it; on the other hand, she allowed it to remain for a considerable time under the left nostril, and even snuffed it up strongly before she was inclined to remove it. During these experiments, we observed that the right eye became suffused with tears; the left eye, on the contrary, appeared to be dry on its surface.

"In order to ascertain further to what degree her sense of smelling was affected, we tried the effect of some substances which possess odor without pungency. On applying oil of anise-seed to her left nostril, while the right one was shut, she inhaled it powerfully, but was sensible of no smell. Then a piece of asafœtida was tried, but still she had no kind of sensation, either pleasant or the reverse. She was sensible to these odors in her right nostril.

"The state of her mouth was examined; with the point of a pencil we pressed against the upper gums, on the left side of her mouth, and the inside of her cheek, where it is reflected off the gums, and she appeared to have very slight or no sensation at all. She volunteered to put a spoonful of mustard between her gums and her cheek, and she seemed very little incommoded by such an experiment. The sensibility of the other parts of her mouth was natural.

"The circumstances of this case," continues Sir Charles, "make it difficult to determine exactly where the disease is seated, which thus produces the destruction of the optic nerve, the third and fourth nerves, the first and second divisions of the fifth nerve, and the sixth nerve. Among these nerves we might add the olfactory nerve; but it may be a question whether the function of that nerve is directly or indirectly affected: the issue of the case will probably determine this matter. However, from the condition of the parts without the orbit, we observe that the power of closing the eyelid and winking is retained, when the power of raising the eyelid is gone, and the sensibility of the eyelids and of the eye itself is completely lost. It is the portio dura which is distributed to the orbicular muscle of the eyelid, and bestows the power of winking. We see also that she can inhale powerfully, and can perfectly move the muscles belonging to the nostril and upper lip of the left side, when at the same time the skin which covers these parts is insensible. Still, that power belongs to the portio dura. This nerve, passing to the face by a circuitous way, and being, therefore, uninjured by pressure within the orbit, permits her to move the left nostril and side of her mouth in a natural correspondence with the other side of her face, although both the first and second divisions of the fifth nerve are included in the disease, and are destroyed along with the first, second, third, fourth, and sixth nerves.

"*May* 20, 1829.—Since she left the hospital she has been a constant sufferer. The pain in her head has never left her; it is principally seated over both her eyes, and over the left in particular. For three years she has observed that this pain is aggravated for a fortnight before her monthly periodical return; she says she does not know what to do, her suffering is so great. The pain varies in a remarkable manner with the changes of the weather: she knows when rain is approaching by the increase of the pain, and immediately after it is over the pain is relieved. She has not had a return of the loss of speech, or of the paralysis of her arm, since she left the hospital, but she has had fits and she has suffered from cramps in the back of her neck and right breast. The arm, which was formerly paralytic, becomes, about once a month, numbed in such a manner that she cannot use her fingers, and this is accompanied with great pain. These attacks do not last for more than five minutes. She walks quite well.

"The loss of sensation is principally in the forehead; when pricked with a sharp point in any part as high up as the crown of the head, she has no feeling; but in the temples, and below the orbits, and on the nose, she retains sensation. The left eye is blind; the pupil large and immovable; the motions of it are gone; the surface is insensible; it is clear, and it remains fixed in the centre of the orbit."

This woman entered the Middlesex Hospital in October, 1824. In the third edition of Sir Charles Bell's work, published in 1844, the foregoing particulars are given, and the history is resumed by Mr. Shaw, as he observed her in June, 1836. At this time there was no marked change, except that, from an inflammation of the right eye, she had lost the sight, and had become entirely blind.

That this case was not one of disease within the orbit is sufficiently apparent from a consideration of the symptoms, almost all of which point to intra-cranial lesion. The extensive paralysis of motion and of sensibility, the epileptic convulsions, the cramps, the aphasia, are so many circumstances against the correctness of Sir Charles Bell's diagnosis. That the morbid condition was inflammation of the basilar surface of the cerebral membranes is extremely probable, as much so upon the principle of exclusion as from a consideration of the positive symptoms.

In a case which I saw in consultation with Dr. H. Knapp, of this city, the patient, a young man, in whom there was no history or even suspicion of syphilis, was attacked with severe pain in the head, attended with dimness of vision in both eyes. In the next place the third pair of nerves became involved, causing paralysis of all the ocular muscles supplied by these nerves on both sides, and of both eyelids, and also producing dilatation of both pupils. Next both fourth nerves were affected; then the fifth pair causing facial anæsthesia and paralysis of the temporal and masseter muscles on both sides; then the sixth, and eventually the seventh and eighth, resulting in paralysis of both external recti muscles, double facial paralysis, and loss of hearing in both ears. There was, therefore, in this very remarkable case, a gradual advance of the morbid process, through a period of several weeks, along the base of the brain, from the anterior to the posterior region. With all these symptoms there was not the slightest mental derangement; neither was there paralysis of any other muscles than of those supplied by the nerves specified. Shortly after I saw him the pneumogastric nerves became implicated, and death soon ensued. Unfortunately, there was no post-mortem examination, but Prof. Knapp and myself agreed that the case was one of inflammation of the membranes covering the basilar surface of the brain.

In the case of a woman who came to my clinique in the winter of 1871–'72, the principal symptoms were deep-seated pains in the head, vertigo, and paralysis of the third nerve on the left side, as evidenced by ptosis, dilatation of the pupil, and external strabismus, the latter condition producing diplopia. Conjoined with these symptoms there was slight but decided paralysis of the muscles of the face, arm, and leg of the opposite side, together with cutaneous anæsthesia. Inquiry showed that these symptoms had been of very gradual development. There was no history of syphilis in the case. I was of the opinion that the disease was chronic basilar meningitis, and gave an unfavorable prognosis; prescribing, however, the iodide of potassium in large doses.

The following year she returned, but this time the sixth nerve was affected, causing internal strabismus; and the ptosis, paralysis of the internal rectus, and the dilatation of the pupil, had entirely disappeared. The other symptoms had for a time been very greatly relieved by the treatment, but had reappeared in considerable intensity about two months previously.

In another instance, this migratory character of the disease was well shown. The case was that of a young man, a private patient, but whom I showed to the class attending my clinique. He came to me originally with external strabismus, ptosis, and dilatation of the pupi of the left eye, together with defective accommodation. Examinatio with the ophthalmoscope showed the existence of optic neuritis, rathe slight in character, but yet decided, in both eyes. He had also the mo tensely agonizing pain in the head that has ever come under my obrvation, with vertigo, frequent attacks of vomiting, and paresis' if t paralysis of the left arm and leg. A consideration of his condition d me to the diagnosis of a cerebral tumor, and I accordingly gave a ry unfavorable prognosis. I was led to this conclusion not so much om the motorial derangement, as from the atrocious cephalalgia from hich the patient suffered. In this case there was some slight suspion of syphilis, and I treated him with mercury and large doses of the dide of potassium. In a short time the pain in his head disappeared, d in a few weeks there were no indications of paralysis anywhere; in ct, he was to all appearances perfectly cured. But at the end of two r three months he reappeared, with the corresponding set of symptoms the right eye and right side of the body, and with pain in the head ally as severe as that which had characterized the previous attack. I gain treated him with mercury and the iodide of potassium, and his ymptoms again disappeared. He remained well for two years, when e had another attack, of which he was entirely relieved by the iodide f potassium.

In this case, the history of which points strongly to a syphilitic rigin, there were probably inflammation and thickening of the memranes at the base of the brain, and presumably gummy formations.

The fact that the inflammation sometimes alternates with skin-erupions is interesting, and has been repeatedly noted. A case of the ind was not long since under my care. It was that of a gentleman ho had attacks of acute pain in the head, accompanied with all the henomena of paralysis of the left third nerve. There was effusion of ymph upon both optic disks, the result probably of old optic neuritis. uriously enough, these attacks alternated with an eczematous affecion, involving the trunk and especially the breast. On the disappearnce of the skin-disease under remedial measures, his head-symptoms mmediately recurred, and, when they were relieved by the action of the odide of potassium, he was again attacked with eczema.

Of the forty-seven cases of basilar meningitis collected by Gintrac,[1] everal of them were distinctly chronic in character. As post-mortem xaminations were made in these cases, they will be more appropriately onsidered under the head of morbid anatomy and pathology.

Causes.—The causes of chronic basilar meningitis are generally ufficiently apparent. It may result from an acute attack, but this not a usual mode of origin, for the reason already stated, that death ordinarily the consequence of such an affection. The most common ause in my experience is syphilis; next, the inordinate use of alcoholic quors; and next excessive emotional disturbance, such for instance as siness anxieties. Then next in point of frequency come atmospheric cissitudes, blows on the head, and attacks of other diseases, as scarlet fever, and especially epidemic cerebro-spinal meningitis, and suppurative otitis. Men are more subject to it than women, and adults more than children. Frequently no cause can be assigned.

---

[1] *Op. cit.*, tome ii., p. 677.

Diagnosis.—Chronic basilar meningitis is not liable to be confounded with any other cerebral affection except tumors, especially those of a syphilitic character, situated at the base of the brain, and chronic softening, arising from thrombosis of the basilar arteries, and diseases of the capillaries.

From non-syphilitic tumors it may be distinguished by the fact that the paralysis is less extensive, that the pain is not usually so severe, that the vertigo is not so intense or persistent, and that the disturbances of vision are not so profound. In a word, the symptoms of chronic basilar meningitis are less pronounced than those of tumors at the base of the brain, while at the same time they are ordinarily developed with greater rapidity. Another mark of difference is the fact that tumors, non-syphilitic in character, do not yield to remedial measures, while chronic basilar meningitis often does, and is generally mitigated by proper treatment.

From tumors of a syphilitic nature, or gummata, as they are called, the diagnosis is difficult, if in fact there is any real distinction existing between them and basilar meningitis of syphilitic origin. A gummy tumor situated at the base of the brain can scarcely exist without the production of basilar meningitis, so that the symptoms such as have been described, present in a person having the clinical history of syphilis, are either the result of simple chronic meningitis, or of meningitis, associated with one or more gummy tumors. Virchow[1] goes so far as to doubt if even, where after death we find only meningitis, the condition has not been preceded by a gummatous affection which has disappeared. The further consideration of this point will be more proper under the head of morbid anatomy.

Where there is no history of syphilis, of course the question of the existence or non-existence of syphilitic tumors will not arise.

From thrombosis of the arteries at the base of the brain, and from such diseases of the capillaries in the same situation as have been described in the previous chapter, chronic basilar meningitis is scarcely distinguishable during the life of the patient. When these are syphilitic in character, the two conditions generally coexist. Sooner or later, however, the former affections terminate in death, and the phenomena to which they give rise, though sometimes remitting in violence, are clearly not lessened in severity by medical treatment. As regards other affections, the history of the case will generally be a sufficient guide to a correct diagnosis.

Prognosis.—The prognosis is very much influenced by the etiology. Those cases which result from injuries generally terminate fatally, as do those due to the excessive use of alcoholic liquors, especially if the habit be continued. When induced by mental influences the prognosis is generally more favorable, provided the patient can be subjected to the hygienic operation of rest, travel, change of associations, etc. Syphilitic basilar meningitis, if seen sufficiently early and subjected to proper treatment, usually terminates in recovery. Subsequent attacks, which are always liable to occur, do not in general run so favorable a course. In all cases a great deal depends upon the duration of the disease. When of long standing the morbid changes in the tissues involved have usually become so profound that recovery is not a probable sequence.

---

[1] "Pathologie des tumeurs, traduit de l'Allemand," Paris, 1869, tome ii., p. 440.

The age of the patient is likewise an important point in the prognosis; and, other things being equal, individuals of advanced years are not so apt to recover as those of middle life. In children a fatal termination is to be expected.

Those cases which are due to the extension of inflammation from the ear almost invariably end in death, as do those ensuing upon epidemic cerebro-spinal meningitis. Latterly, however, I have had under my charge two cases, resulting from cerebro-spinal meningitis, in which it has taken place, though with very marked impairment of vision from double optic neuritis in both, and of hearing in one.

Morbid Anatomy.—The morbid anatomy of chronic basilar meningitis does not differ in many respects from the corresponding affection of the convex surface of the brain. It is, however, generally much more circumscribed in its extent, and may be restricted to a portion of the membranes not larger than a dime in circumference. In one form the affected tissues are thickened and opaque, and there is an exudation of serous or gelatiniform fluid; in another the exudation is puriform; and in a third it is thick and gummy, constituting the so-called gummy tumor of syphilitic origin.

The serous or gelatiniform exudation often shows a tendency to become organized and to present a membraniform appearance, or even to assume a still more solid form. Gintrac cites from Simon[1] the case of a woman, thirty-five years old, who for six years had been subject to paroxysms of intense cephalalgia. Two years subsequently she became blind on the left side, and for two months afterward suffered still more severely from pain in the head; then she lost the sight of her right eye. Both irides remained contractile. The sense of smell was lost, though the pituitary membrane retained its tactile sensibility. Hearing, taste, and touch, were unaffected. Coma supervened, in which she died. On examination, the diploë and the membranes were found congested. The arachnoid and the ventricles contained an excess of serous exudation. In the pia mater there was a deposit of a whitish-gray fibrinous substance which followed the course of the middle cerebral vessels, and lay over the chiasma of the optic nerves, the tubercula mammillaria, and the anterior perforated spaces. The optic and olfactory nerves were atrophied and the chiasma deformed; the retinæ were normal.

---

[1] "Bulletin de la société anatomique," 1845, p. 196.

Usually the membranes are, in some places, firmly adherent to each other, and not infrequently to the cortical substance of the brain, in which case the latter is softened to such an extent as to tear away when the attempt is made to separate the membranes from it.

When the exudation is puriform in character it occasionally becomes thick, and appears as semi-solidified plates in various situations.

The exudation, whatever its nature, may be deposited between the layers of the arachnoid, in the sub-arachnoid space, or in the meshes of the pia mater. Its seat may be any part of the base of the brain, but its usual situations are the chiasma of the optic nerves, along the course of these nerves, on the tuber cinereum, the corpora mammillaria, and between the crura cerebri. Sometimes it extends anteriorly along the course of the olfactory nerves, laterally into the fissure of Sylvius, and posteriorly as far as the pons Varolii and medulla oblongata.

In the syphilitic form of the disease it is a matter of some doubt whether the gummy exudation is the result of the specific inflammation of the membranes or whether the inflammation is excited by the presence of the new formation. Gintrac[1] seems inclined to doubt the existence of syphilitic meningitis, though he admits the possibility of its occurrence. For him there is no syphilitic meningitis unless its presence be demonstrated by a post-mortem examination and its characteristics definitely established, while others give a specific nature to any inflammation of the meninges—and, in fact, to any other affection—occurring in a person who, at any time, has been the subject of syphilis. In my opinion, cerebral meningitis may be induced by the syphilitic diathesis, and thus be a syphilitic meningitis, and it may exist as a non-specific affection in an individual who has had an infecting chancre. Undoubtedly there are cases of meningitis occurring in syphilitic persons that are no more under the influence of anti-syphilitic treatment than the cases happening in otherwise healthy individuals. Fox,[2] however, states it as his opinion that it is at best an open question whether meningitis ever occurs independently of syphilis, rheumatism, alcoholic poisoning, tubercle, anæmia, or mechanical irritations.

But, in regard to the morbid anatomy of chronic basilar meningitis of syphilitic etiology, Virchow[3] has supplied very important data in his remarks on syphilitic tumors of the brain and its membranes.

The gummy tumors are seen most frequently at the base of the brain. Sometimes they are very exactly defined in their boundaries, and then they are tumors in the true sense of the word; but ordinarily they are more diffused, and are accompanied with the phenomena of inflammation, a fact which seems to distinguish them from the true tumor. As already stated, Virchow regards this condition as a "gummy inflammation;" and even when the exudation is not present, and the appearances are those of a non-specific inflammation of the membranes, the question may arise whether or not the gummy exudation has not been the first step in the morbid process, but, having been absorbed, has left only doubtful traces of its presence. With the true gummy tumor we are not at present concerned.

---

[1] *Op. cit.*, tome iii., p. 100.

[2] "The Pathological Anatomy of the Nervous Centres," London, 1874, p. 65.

[3] *Op. cit.*, p. 437, *et seq.*

The most common seat of syphilitic basilar meningitis is the region bounded anteriorly by the chiasma of the optic nerves, and posteriorly by the crura of the cerebellum. Hence it is that the nerves lying at the base of the brain, and especially the third pair, are so liable to be implicated. This latter, from its exposed situation, running as it does from the crura cerebri to the orbit, can scarcely escape being involved in the morbid process.

Pathology.—The functions of the nerves at the base of the brain are so well understood that the connection of the symptoms of chronic basilar meningitis with the morbid condition constituting the disease is sufficiently apparent in the great majority of cases. The circumscribed character of the inflammation enables us also to determine its seat with accuracy, and its migrations can be marked with considerable certainty. Probably in the very earliest stage of the disease these points cannot always be clearly made out, for the principal phenomenon is centric pain, due to congestion, and it is difficult to locate the seat with exactness; but, as the affection advances to its full development, effusion takes place, and then the eccentric symptoms become more prominent if they do not at this time make their appearance. These we have seen consist of disturbances of sensibility and of motility in those parts of the body supplied by the nerves at the base of the brain, or of aphasia from the extension of the inflammation along the fissure of Sylvius to the island of Reil, or parts of the brain in its immediate vicinity. It is only at a still later period, when the morbid process has directly or indirectly involved the substance of the basilar surface of the brain itself, that sensibility or motility is disturbed in the trunk and limbs.

When the sense of smell is deranged, the lesion exists upon the same side as the symptoms, for, as we know, the olfactory nerves do not decussate.

When vision is impaired from optic neuritis, we cannot be so sure as to the side upon which the disease exists. For we may have optic neuritis as the consequence of disease in distant parts of the brain, as well as from the direct implication of the optic nerves in the pathological condition; and even when this latter is the case, owing to the incomplete decussation of these nerves, it is possible for optic neuritis to exist in conjunction with a homolateral or a heterolateral lesion.

The symptoms due to the involution of the third pair of nerves are manifested as regards the upper eyelid, which becomes paralyzed and drops over the eye, the muscles of the globe, except the external rectus, and the pupil, which is dilated, owing to the paralysis of the circular fibres of the iris, which receive their motor influence, through the third nerve, from the ophthalmic ganglion.

The third pair of nerves have their apparent origin in the crura cerebri, the right nerve from the right crus, and the left nerve from the left crus. If, however, the fibres be followed out by minute dissection, as has been done by Vulpian[1] and others, they are seen to be arranged into three groups. Of these the middle and posterior decussate after passing entirely through the crus, while the anterior group passes forward to the optic thalamus, in which ganglion the fibres are lost. None of the fibres of origin originate in the crus, and this latter may be entirely dissected away and the third nerve be left intact. Now, if Vulpian be right in his view—and there is no doubt now upon the subject—that a considerable number of the fibres of origin of the third nerve decussate, any disease of the brain affecting these fibres must be manifested by derangements of motility of the muscles supplied by the nerve of the opposite side, and, as the motor and sensory fibres of the spinal cord decussate below the point at which the third nerves decussate, the disease, if causing paralysis of other parts of the body, would induce this condition also on the opposite side; or, in other words, on the same side with the paralysis of the muscles supplied by the third nerve. Each crus cerebri contains the motor and sensory fibres which come from the opposite side of the body, below the medulla oblongata. It also contains the fibres of the corresponding third nerve. Disease involving a crus would therefore cause derangement of motility in the muscles supplied by the corresponding third nerve, and of sensation and motion in the opposite half of the body; alternate or cross paralysis would therefore be the result. As chronic basilar meningitis often involves the membrane covering a crus, cross-paralysis is frequently a phenomenon of the disease.

In those cases in which there is no paralysis anywhere except in the muscles supplied by the ocular motor nerve, the lesion must exist anteriorly to the crus, and affect the trunk of the nerve of the same side as that of the paralyzed muscles.

It is not often the case that the fourth nerve, or trochlearis, is alone involved; though one such case has come under my observation. In this the patient had no marked symptom of any kind, except that in a certain position of his head he saw double. On examination, I ascertained that, when he turned his head toward the left shoulder, he saw double, and hence I diagnosticated paralysis of the left superior oblique muscle. Further experiments confirmed this opinion, and the diagnosis of chronic basilar meningitis was shown to be correct by the extension of the disease so as to involve the third nerve, and by the supervention of pain and other phenomena of the affection in question.

---

[1] "Essai sur l'origine de plusieurs paires des nerfs craniens." Thèse de Paris, 1853.

The sixth nerve, or abducens, is not infrequently the only nerve implicated in the lesion, and then there is internal strabismus from paralysis of the external rectus muscle. Several such cases, in which there were the concomitant symptoms of chronic basilar meningitis, have come under my notice. The case of one of these, a woman, who formed the subject of a clinical lecture, has already been cited. Another case was that of a man, the subject of syphilis, and in whom the lesion was only manifested as regarded the external rectus muscle. There were no head-symptoms of any kind. The paralysis had ensued during the night, and the patient awoke in the morning to find that he had internal strabismus and double vision. He recovered entirely under the use of large doses of the iodide of potassium. But on the 10th of January, 1875, he had an epileptiform paroxysm, and this was several times repeated during the following week. Under the influence of the iodide of potassium, conjoined with the bromide, he has for the past two months had no return of the convulsions; but his mind is somewhat confused, and he has occasional severe pain in the head.

The seventh, or facial nerve, is sometimes embraced in the morbid process, giving rise to paralysis of one or more of the muscles on one side of the face, which it supplies. In one instance, apparently the result of syphilitic basilar meningitis, which came under my charge in December, 1874, both facial nerves were involved, and there was consequently double facial paralysis.

The eighth, or auditory nerve, also occasionally gives evidence of loss or impairment of its function; but, unless special examination relative to the hearing be made, or both nerves be involved, the lesion, as regards this nerve, may escape detection, as patients very often, even when the hearing is entirely destroyed in one ear, are unaware of the fact, and persist that it is unimpaired.

The ninth, tenth, and eleventh pairs of nerves are not so apt to be affected in chronic basilar meningitis as some of the others, for the reasons that their relations with the interior of the cranium are not so intimate, and that the seat of the disease is generally anterior to their situation.

Should the ninth, or glosso-pharyngeal nerve, be involved, there would be loss or impairment of the sense of taste upon the corresponding side, and the implication of the pneumogastric would lead to a complicated series of phenomena, of which the chief would be palpitation of the heart, irregularity of the respiration, and derangement of the function of digestion; while, if the spinal accessory were reached by the morbid process, there would be difficulty of swallowing, and perhaps alteration in the timbre of the voice.

The hypoglossal, or sublingual nerve, is occasionally affected, producing paralysis of the side of the tongue corresponding to the situation of the disease.

When the fifth nerve is involved, the chief manifestations of its lesion are relative to sensation. Thus there are either intense neuralgic pains in some part of the cutaneous surface of the head or neck, or there is equally well-marked anæsthesia. The former condition is by far the more frequent. From some cause or other, the motor fibres of this nerve almost invariably escape, and thus the temporal and masseter muscles are not paralyzed. I have, however, already cited a case in which they were affected.

The general relation of the symptoms of chronic basilar meningitis with the lesion constituting the disease is well shown in several of the cases cited by Gintrac. Thus he quotes one from Bossu,[1] that of a man, twenty-four years old, who from exposure became affected with headache about the supra-orbital region, vertigo, noises in the ears, facial neuralgia, and muscular contractions. At the end of a year he had vomiting, want of appetite, general debility, and a continuation of the supra-orbital headache. There were also amblyopia, diplopia, external strabismus, dilatation of the pupils, and painful contractions of the right side of the face. The pulse was full, regular, and not frequent; the mind was unaffected. Coma supervened, the right side of the face became insensible, the evacuations were involuntary, speech was impossible, and the movements of the tongue were imperfectly performed. The pulse was feeble and frequent, and death ensued. On post-mortem examination, a reddish serum was found to be infiltrated between the convolutions. At the base, under the third ventricle, a gelatiniform substance enveloped the commissure of the optic nerves and the tuber cinereum. It was reddish in color, and was closely adherent to the pituitary gland. The tubercula mammillaria were separated by a reddish mass, which extended into the ventricle, and which there had the size and form of a nut.

The following case, cited by Gintrac[2] from Simon, is equally interesting:

"A woman, thirty-five years old. For six years accessions of pain in the head. Two years afterward blindness of the left eye, and then for two months the most intense cephalalgia, followed by loss of sight in the right eye. Pupils still active. Anosmia, although the pituitary membrane preserved its tactile sensibility. Hearing, touch, and taste unimpaired. Skin warm; pulse freqnent, hard, and small. Failure of appetite; thirst, constipation, coma, death.

"There was congestion of the diplöe and of the meninges. The arachnoid and the lateral ventricles contained serum. There was a grayish-white deposit, of fibrinous appearance, in the pia mater, along the course of the middle cerebral vessels, on the chiasma of the optic nerves, the tubercula mammillaria, and the anterior perforated spaces. The olfactory and optic nerves were atrophied, and the chiasma was deformed. The retinæ were normal. The tissue of the brain at the base was superficially softened."

---

[1] *Gazette médicale de Lyons et moniteur des hôpitaux*, 1855, p. 853.

[2] "Bulletin de la société anatomique," 1860, p. 143.

Treatment.—The principles which have been laid down for the management of cases of chronic verticalar meningitis are equally applicable to the basilar form of the disease. The iodide of potassium, conjoined with some one of the bromides, should be administered; and, in syphilitic cases, the former should be pushed to its extreme limit by gradually increasing the doses. At the same time, there are other means of treatment, which are rendered necessary by the existence of paralysis, and these ordinarily consist of strychnia and some form of electricity. The details will, perhaps, be more clearly shown by the citation of a few cases from my note-book:

A. W., married, aged thirty-two, consulted me, April 7, 1873, for pain in the head, accompanied by paralysis of the third nerve on the left side, producing ptosis, external strabismus, dilatation of the pupil, and double vision. On examination with the ophthalmoscope, both optic papillæ were found to be congested, the left far more so, however, than the right. He had had an epileptiform convulsion about two weeks before coming to me, and had suffered very often from attacks of vertigo. The first evidence of the disease was the cephalalgia, which had been very gradually developed during six or seven months, and which was mainly confined to the left temporal region. The paralysis of the third nerve had been suddenly produced, on the morning of the 1st of April, while he was eating his breakfast.

There was not the least evidence of syphilis in this case. The affection had obviously originated from long-continued anxiety of mind, the consequence of business troubles.

I immediately began the administration of the iodide of potassium in the form of the saturated solution, in doses of ten drops three times a day, increased to twelve drops the second day, fourteen the third, and so on. After the fourth day, the intense pain in the head began to diminish; and on the tenth day, when the patient was taking thirty drops—equivalent to thirty grains—three times daily, it entirely disappeared. The paralysis of the third nerve, however, continued, although the doses of the iodide were carried up to over two hundred grains daily, or seventy grains at a dose. The medicine was then discontinued, and the patient was treated with gradually-increasing doses of strychnia, and the interrupted primary or galvanic current applied to the closed eye, as nearly as possible over the internal rectus muscle on the upper eyelid. This treatment was persevered with for several weeks, without any marked effect upon the paralysis of the upper eyelid, though the internal rectus muscle gradually recovered its power, and the diplopia disappeared. Nearly a year afterward, when I aga saw the patient, the lid still drooped; but there had been no return the other symptoms.

A gentleman, aged about fifty, single, consulted me on the 11th August, 1874, for intense pain in the right side of the head, wi which he had suffered for several months, night and day. Upon exam ination, I discovered that he had experienced an attack of iritis of t left eye ten years previously, and that there was other evidence syphilis. There was paralysis of the internal rectus of that side, whic caused strabismus, though no diplopia, as the sight of the eye had been lost by extension of the inflammation to the capsule of the lens, causing opacity. In conversation with him, I observed that he was deaf in the right ear, a fact which he had not noticed till his attention was called to it and the hearing capacity tested. On examining the ear with the speculum, I perceived that the external auditory canal was closed by a growth of some kind, which was adherent to the anterior wall. The ophthalmoscope revealed the existence of marked optic neuritis of the right eye, and the patient could not read No. 3 of Galezowski. No examination could be made of the left eye.

On the following day, when he made his visit to me, the right side of his face was paralyzed, as was also the right side of the tongue, and his speech was, in consequence, rendered very difficult and indistinct. I then began the administration of the iodide of potassium, in the form of the saturated solution, starting with the dose of ten drops three times a day, and directing it to be gradually increased. This was continued till the 14th, when I removed the growth from the ear, by excision, with a delicate bistoury. The effect of this operation was at once evident, so far as the hearing was concerned, and the patient declared that the pain in the head was decidedly mitigated. As it still, however, continued, I augmented the doses of the iodide by six drops a day, instead of three, and began the application of the interrupted primary current to the paralyzed muscles of the face and tongue. On the 20th he was taking twenty-one grains three times a day. The pain was decidedly less; but, as there were sharp lancinating pains along the course of the auricular branch of the lesser occipital nerve, I made an incision through the scalp, so as to divide it. The effect was, to abolish this pain altogether. The intra-cranial pain gradually diminished under the increasing doses of the iodide, and on the 27th of August had entirely ceased. The medicine was continued for several days afterward, and was then omitted. The tongue gradually improved in motor power; but several months subsequently was not protruded straight, although the speech was as good as ever. There has been no return of the other symptoms.

The growth removed from the ear was examined microscopically by my friends Prof. Roosa and T. E. Clark, as well as by myself, and we agreed in the opinion that it was neuromatous in character. The whole tumor was somewhat larger than a large pea.

The following very interesting case, which occurred recently in my practice, I quote from Dr. Lente's excellent paper "On the Neurotic Origin of Disease,"[1] read before the New York Neurological Society, December 7, 1874. Dr. Lente had frequent opportunities of seeing this patient in my consulting-room, and of witnessing the results of the treatment. Mr. W. was also kind enough to allow me to present him at my clinique at the Medical Department of the University of New York, and to describe his case to the class in attendance.

"The treatment of the following case I had the opportunity of watching, through the courtesy of Prof. Hammond. The history I had from the patient himself:

"Mr. W., a grain-inspector of Chicago, was attacked three years ago with epileptic convulsions; has had them once a month or oftener; also some threatening cerebral symptoms; had no treatment that he knows of except moderate doses of bromide of potassium and chloroform inhalation. In June last he had a recurrence of cerebral symptoms, insomnia, pain, double vision, etc. This lasted two weeks, and disappeared. On the 14th July, after some exposure to the sun, he was again attacked with the above symptoms, to a greater degree, and with complete inability to raise the eyeball or upper eyelid (left eye), also extreme internal strabismus, diplopia, and severe cephalalgia. These symptoms occurred suddenly in the night. Could neither read, nor distinguish the quality of grain. The strabismus disappeared slowly, and the ptosis also diminished somewhat, so that when he applied to Dr. H., about the 13th of October, 1874, he could, *by an effort*, raise the lid so as to expose the cornea, but it fell back immediately; other symptoms the same. He was put upon increasing doses of the iodide of potassium, with the idea of relieving the *basilar meningitis*, presumed to be the cause of the symptoms, the application of the induced current to the brow and temple, and the hypodermic injection of strychnia. No *immediate* effect could be expected from the first two remedies; it is to the last that I desire to direct attention. Prof. Hammond proposed to inject the solution directly into the affected muscles, and accordingly did so, using gr. $\frac{1}{32}$ in two drops of water; it is presumed that it passed into the muscle, or most likely in its immediate proximity. In all, six injections, I think, were used. I watched the effect carefully and tested the eye and lid after each. They were done each alternate day. He declared that he perceived quite a decided effect. After the second there was no doubt, as I could see the change within fifteen minutes, both on the ball and on the lid, but especially on the latter; after the third, the *ptosis* had entirely disappeared, and he could raise the *ball* to an horizontal plane; the diplopia had disappeared, and he could read by holding the book low. After the fifth injection (gr. $\frac{1}{20}$) no difference in the appearance of the eyes was distinguishable, and he could read with the book held directly before him. He considered himself cured."

---

[1] *Psychological and Medico-Legal Journal*, February, 1875, p. 82.

In this case the iodide was carried to doses of sixty grains three times a day, before the pain began to yield; and eighty grains, equal to two hundred and forty grains daily, was reached, and continued for several days, before it was deemed advisable to omit its use.

Mr. B. was sent to me, December 19, 1874, by Prof. M. A. Pallen. At the time he was suffering from agonizing pain in the left side of the head, paresis of the whole right side of the body, except the face, aphasia, of the amnesic variety mainly, although the power to coördinate the muscles of articulation was greatly impaired, and from decided mental disturbance, characterized by the existence of hallucinations and marked dementia. The sight of both eyes was weakened, and examination with the ophthalmoscope showed the existence of double optic neuritis. There was a clear history of syphilis.

I immediately began the administration of the iodide of potassium, in ten-grain doses, three times a day, gradually increased, as in the foregoing cases. By the time twenty-grain doses were reached the pain in the head had disappeared, the speech was much improved, the weakness of the right side had diminished, and the mind was altogether stronger. The iodide was continued up to sixty-grain doses, and then, as the patient was apparently cured, it was omitted, and he resumed his duties as cashier in a bank.

Two months afterward, he had a relapse into his former condition. The accession was sudden. He awoke in the morning with pain in the head, weakness of the right side, and complete loss of speech. His aphasia was removed by a single application of the galvanic current from ten cells to the tongue, and I increased the use of the iodide as before. He again recovered his health. He is now (March 23d) quite well.

It would be very easy to adduce many other cases from my private and hospital practice, but the foregoing are sufficient to indicate the main principles of treatment in chronic basilar meningitis. Occasionally, in cases of syphilitic origin, in which the infection has been recent, it may be advisable to administer mercury in some one of its forms. The bichloride, in the dose of the one-sixteenth of a grain, may be given with each dose of the iodide of potassium, or the biniodide in like doses, in the form of pill. Whether the affection has a syphilitic origin or not, antiphlogistic measures, as they are called, are not proper. On the contrary, wine and highly-nutritious food are frequently productive of amelioration.

Should insomnia be present, some one of the bromides should be given, in doses of from fifteen to thirty grains, three times a day, till its full effect be produced.

It may be stated that I have never observed any ill effects follow he administration of the very large doses of the iodide of potassium which I have recommended. Coryza is certainly not more apt to occur han with the small doses, nor is it more severe. Gastric irritation can generally be prevented by diluting each dose in a sufficient quantity of water. A dose of fifty or sixty grains should never be taken in less han half a tumbler of water.

In the treatment of the paralysis which often remains, even after all active disease within the cranium has disappeared, electricity is almost indispensable; and I am entirely satisfied that the hypodermic injection of strychnia into the paralyzed muscle, or as near as may be to it, is a measure of the utmost importance. The good effects of it were very clearly seen in one of the cases cited.

CHAPTER X.

TUBERCULAR CEREBRAL MENINGITIS.

INFLAMMATION of the membranes of the brain, attended with or due to a deposit of miliary tubercles, was for many years considered as a disease peculiar to infancy, and was known as acute hydrocephalus before its morbid anatomy and pathology were clearly comprehended. It is now well understood to be an affection to which adults are liable.

By some authors, especially Robin and Bouchut, it is regarded as not being tubercular in character. It has hence occasionally been termed granular meningitis. Although mentioned by the ancient medical writers, no clear and systematic description of tubercular meningitis was given till Whytt[1] published his essay on the subject of dropsy of the brain. Since that time it has received the attention of many writers in this country, Great Britain, France, and Germany.

Symptoms.—Whytt defined three periods of the disease, which he marked by the state of the pulse. I think the symptoms may be properly arranged in four stages: 1. The prodromatic stage; 2. The stage of excitement; 3. The stage of depression; and 4. The stage of recurrence.

1. THE PRODROMATIC STAGE.—This period may be altogether wanting, or may be so slightly manifested as not to be noticed. Generally, however, it is well marked.

If the child be sufficiently advanced in years, a change of disposition is among the first symptoms perceived. Thus the temper becomes irritable, caresses are disregarded, and dislike is shown for those amusements which formerly gave pleasure. At the same time the appetite disappears, and the child loses flesh rapidly. This latter is not noticed about the face, but is mainly confined to the abdomen and limbs. The bowels are generally obstinately constipated, but occasionally there is diarrhœa. Headache is not often complained of; neither is vomiting a common symptom of this period. Fever is not continuous, although it is generally present at irregular times of the day.

---

[1] "Observations on the most Frequent Form of the Hydrocephalus Internus, viz., Dropsy of the Ventricles of the Brain. Works of Robert Whytt, edited by his Son." Edinburgh, 1768, p. 725.

The prodromatic stage may last only a few days, or may be prolonged for two or three months.

2. THE STAGE OF EXCITEMENT.—This period is ushered in by obstinate vomiting, which is present in many cases, even though no food be taken. Intense pain in the head is a coincident symptom, and is so severe that the child puts his hands to his head and cries out or awakes screaming. Convulsions may also occur. They do not differ in general appearance from the ordinary epileptic paroxysms, and may be repeated several times.

Very early in this stage the fever becomes more persistent than in the first stage, although it may still be irregular. The pulse, however, is not hard and resisting, as in other inflammatory affections, but is soft and compressible.

Trousseau[1] has called attention to a condition of the skin present in tubercular meningitis, which he at first regarded as peculiar to this disease, but which subsequent investigation showed was likewise found in simple meningitis, in typhoid fever, and some other affections. If the finger-nail be passed lightly over the surface of the abdomen or the thorax so as to trace a series of lines, in about thirty seconds the skin becomes red—the color being at first diffused, but very soon the lines made by the nail are indicated by a still redder color, which persists a long time. Trousseau calls this appearance the "cerebral stain" (*tache cérébrale*). The phenomenon he attributes to a profound modification in the vascularization of the skin; and, although it is not to be regarded as absolutely pathognomonic, it is a sign of very great importance.

The intellectual faculties are not yet affected to any considerable extent, but the changes of character and disposition, and indifference to things which formerly excited interest, are still well marked.

The physical strength, though lessened, is still not yet so far reduced as to oblige the patient to remain in bed.

The tongue is usually coated and red at the edges, the appetite diminished, and the bowels are obstinately constipated.

The temperature of the body is elevated, but not to an extreme degree; the thermometer indicating from 101° to 103° Fahr. Sometimes there are distinct remissions in the violence of all the symptoms, but the disease nevertheless goes on to its full development. The transmission from the second to the third stage is often marked by an amelioration which may last several days.

---

[1] *Op. cit.*, Leçon lv., "Fièvre Cérébrale."

From what has been said, it will be seen that the characteristic phenomena of this stage are headache and vomiting. Its duration varies from seven to fourteen days.

3. Stage of Depression.—The pulse, which in the previous stage was sometimes as high as 140, and sometimes as low as 80, now becomes less rapid than is normal, and may even fall below 50. At the same time the beat is quick, but the interval between the pulsations is at times so great that the observer is, as Dance[1] says, fearful that the action of the heart has stopped. The interval between the pulsations is often irregular, and this may be regarded as a sign of unfavorable import.

In young infants there is a reduction in the temperature of the body below the normal standard, which lasts throughout the whole of this period. Roger regarded this reduction, preceded as it is by a higher temperature, and followed during the succeeding stage by another elevation, as pathognomonic of tubercular meningitis.

The continued excitement of the previous stage is replaced in this by a strong tendency to somnolence, which alternates with a rather quiet delirium. The patient lies on his back, with the eyes fixed, but yet not looking at any object with attention. Events taking place around him no longer attract notice, and, though when addressed in a loud tone he may turn his gaze toward the speaker, it is very evident that the words convey no idea to his mind.

The fingers are kept in almost continual motion, picking up threads and other small objects from the bedclothes, and occasionally clutching at imaginary things. Again, the fingers are alternately opened and shut without any real or apparent motive, and again the head is turned restlessly from side to side of the pillow. Convulsions are very generally present from time to time during this stage, and may be so frequently repeated as to leave scarcely any interval between the seizures. Even if the attacks do not involve the body generally, the eyes scarcely ever escape; there being strabismus, convulsive movements of the pupils, and constant motions of the eyeballs. The facial muscles are likewise often affected.

In the intervals of wakefulness, the cephalalgia continues, and causes the peculiar scream which is so characteristic as to have received the name of the "hydrocephalic cry." It is a sound such as might be produced by mingled emotions of terror and grief. Although probably excited by the pain, it is more or less automatic, and is not exactly such a cry as would be produced by unmixed physical suffering. It is accompanied, however, by that contraction of the muscles of the face indicative of suffering.

---

[1] "Mémoire sur l'hydrocéphale," *Archives générale des médecine*, 1830.

The paleness of the countenance continues, but at times there is a sudden redness, which disappears as rapidly as it comes.

The conjunctivæ are generally injected, and photophobia is presen M. Bouchut,[1] who has given great attention to the subject of ophth moscopy in diseases of the nervous system, finds peripapillary co gestion, dilatation of the retinal vessels, and deformation of the papill

There is often a general hyperæsthesia of the skin, for which, ho ever, anæsthesia may be substituted. When this latter is the case t conjunctivæ participate, and inflammation results.

The limbs are weak, and, should the patient attempt to walk, the gait is staggering. The speech is hesitating, is rarely indulged in except in response to questions, and then with the least possible expenditure of words.

The vomiting, which formed so prominent a symptom of the previous stage, has ceased, but the constipation still persists.

The respiration is irregular, sometimes being rapid and sometimes slow. Occasionally there are deep sighs, followed by numerous quick inspirations, and again the respiratory movements may be so slight as scarcely to be perceived. This variation from the normal action, as well as the irregularity of the heart's movements, is due to the implication of the pneumogastric nerves at their origins.

This stage may last for from two or three days to two weeks.

4. Stage of Recurrence.—The characteristic phenomena of this stage are the return of the fever and the increase in the violence of the symptoms indicative of cerebral disturbance. Before its onset there may be a period of nearly complete intermission, so that the impression may be formed that recovery is taking place. This apparent cessation of the morbid action, however, only serves, with the experienced observer, to make the reappearance of the symptoms more striking.

Convulsions are more frequent and violent than in the previous stage, and tonic contractions of the limbs are not uncommon. These contractions are more generally met with in the muscles of the neck and upper extremities, and vary from time to time in their intensity. The head is thus thrown backward, and, as the morbid action frequently extends to the muscles of the back, an appearance in the patient not unlike that present in tetanus is produced.

Paralysis eventually supervenes. At first this is incomplete, affecting only a single limb or the muscles of the face, but it extends, and both limbs on one side, or an arm and a leg of opposite sides, become involved. Voluntary power is lost, but reflex movements can be excited by pinching or tickling.

The delirium acquires increased intensity, and alternates with the somnolence, which likewise becomes more profound, and which gradually masks all the other symptoms, till at last the coma is persistent and general, and spinal sensibility is lost.

---

[1] "Du diagnostic des maladies du système nerveux par l'ophthalmoscopie," Paris, 1866, p. 45, *et seq.* Plates iv., v., vi., vii., viii., ix., and xi., of the Atlas.

Before death the pulse rises in frequency, a cold sweat makes its appearance, and the patient dies either by a slow process of asphyxia, or in convulsions.

The fact that tubercular meningitis is not confined to infants is now generally admitted. Dance[1] was the first to recognize its occurrence in adults, and Gerhard,[2] of Philadelphia, a few years subsequently reported several cases. Ledibuder[3] also pointed out the analogy between the tubercular meningitis of infants and that of adults, and still later Valleix[4] gave the weight of his authority to the same effect.

So far as the symptoms are concerned, I have never been able to perceive any essential points of difference between the tubercular meningitis of children and that of adults.

The affection is, of course, modified, as are all other diseases, by the age of the patient, but, when allowance is made for this factor, the morbid process is one and the same in character. In adults, however, it generally supervenes in the course of tuberculosis of the lungs, whereas in infants it is ordinarily a primary manifestation of the tubercular diathesis.

Causes.—Tubercular meningitis is an expression of a general state of the system. To enter at length into the question of its etiology would necessarily involve a discussion of the cause of the diathesis to which it is essentially due. Nevertheless, there are a number of determining causes that may be appropriately considered. Age is an important factor in determining the accession of tubercular meningitis. It is rare during the first year of infancy, but is more common during the period extending from the second to the seventh year than any other time of life. From eight to ten it is much less frequent, and from ten to fifteen is rarely seen.

In adults it is most common between the ages of seventeen and thirty. From thirty to forty it is rare, and after forty is scarcely ever met with.

Males are more frequently the subjects of tubercular meningitis than females, and this holds good for all ages of life.

The season of the year appears to exercise no influence.

As to many other exciting causes alleged by authors, such as blows, emotional excitement, and previous diseases, nothing very definite is known. The same cannot, however, be said of the morbific influence of bad air, insufficient food, improper clothing, neglect of cleanliness, and a disregard of other sanitary requirements.

---

[1] *Op. cit.*

[2] *American Journal of the Medical Sciences*, 1834.

[3] "Essai sur l'affection tuberculeuse aiguë de la pie-mère," Paris, 1837.

[4] "De la méningite tuberculeuse chez l'adult." *Archives générales de médecine*, 1838.

Diagnosis.—Tubercular meningitis is liable to be confounded with several other affections, and can sometimes only be distinguished with difficulty.

From simple meningitis it may be diagnosticated by the facts that the onset of the former is sudden, while the latter is insidious in its approach, and slow in the development of its symptoms; the one goes on steadily through its course, the other halts and remits; in the one the temperature of the body rises several degrees, in the other the elevation is scarcely ever more than two degrees; in the one there is no hereditary tendency, while in the other inquiry will usually reveal the existence of hereditary tubercular predisposition.

The mental symptoms show a marked difference. In simple meningitis the delirium is often furious, and is always very active; in the tubercular form of the disease the delirium is quiet, and alternates with stupor.

In typhoid fever there may be vomiting and headache, but the bowels are not constipated, and there is tenderness over the right hypogastric region. Moreover, the epistaxis, the eruption, and the swelling of the spleen, which occur in typhoid fever, will aid in making the diagnosis more certain.

Worms in the alimentary canal may give rise to a set of symptoms very similar to those which form the prodromata of tubercular meningitis. As Jaccoud observes, therefore, it is well, whenever a child exhibits these symptoms, to administer one or two doses of a strong vermifuge.

A peculiar affection, to which young infants are liable, may be mistaken for tubercular meningitis. It was first described by Dr. Gooch,[1] but derived its name—"hydrocephaloid disease"—from Dr. Marshall Hall. I have already alluded to this disorder under the head of cerebral anæmia. In it the child is irritable, restless, starting at every noise, moving in sleep, and often waking screaming. Vomiting is frequently present, but the bowels are loose. The whole appearance of the child betokens exhaustion, and, if due care be not taken, death may ensue. The absence of constipation, the history of the case, and the depressed state of the fontanelle, if this be yet open, will suffice to render the diagnosis clear.

Trousseau considers the irregularity of the respiration the most important sign indicating the presence of tubercular meningitis. "In no other disease," he says, "will you meet with this singular anomaly. You will not observe this unequal and irregular respiration either in the essential convulsions of infancy or in typhoid fever. I have reason, then, for insisting on the importance of the symptoms."

Prognosis.—There is not much to say under this head. The ordinary termination of the disease is death. I have never seen a case recover; and, though instances with a favorable result have been reported, I am disposed to think the diagnosis of such has been erroneous. Drs. Meigs and Pepper,[1] of thirty-one cases, had no recovery, though they report a case of tuberculosis of the meninges—not tubercular meningitis—in which recovery appears to have taken place, though the child died a year or two afterward with dysentery.

---

[1] "On Some Symptoms in Children erroneously attributed to Congestion of the Brain." Gooch's Essays, New Sydenham Society, 1859, p. 179.

It seems contrary to reason to expect a radical cure in a disease in which the cause cannot be removed. Do what we will, the tubercular deposit remains; and, as Jaccoud remarks, the reported cases of recovery were rather instances of a long remission in the intensity of the symptoms. Seitz,[2] in his recent treatise, asserts that the time when cases of acute hydrocephalus were cured has gone by, and that former apparent success is to be attributed to false diagnosis. He declares that he has never witnessed a case terminate favorably.

Morbid Anatomy and Pathology.—A question arises at the outset of an inquiry relative to the morbid anatomy of tubercular meningitis, which refers to the essential character of the disease; and that is, whether the gray semi-transparent granulations met with on post-mortem examination are tubercles, or whether they are an entirely distinct morbid product? Valleix, Rilliet and Barthez, Barrier, Grisolle, Meigs and Pepper, and others, regard them as tubercles. Grisolle expresses himself clearly on this point. "We have no doubt," he says, "that these granulations are tubercles in a rudimentary state; for we have many times, in the same subject, followed the morbid product in its different phases of evolution from the amorphous condition to the fully-developed tubercle."

On the other hand, Bouchut, basing his conclusions mainly on the microscopical observations of Robin, is of the opinion that the granulations are formed: 1. Of fibro-plastic elements, consisting of free nuclei and fusiform cells, and ovoid cells. The nuclei are ovoid or spherical, and generally very small, not exceeding 0.008 to 0.009 in. in diameter. 2. Of a great quantity of granular amorphous homogeneous matter, which keeps the other elements strongly united. 3. Of a few vessels and fibres of connective tissue. Among all these elements the tubercular corpuscles of micrographers are not to be found; and, therefore, the disease cannot be regarded as tubercular in character. M. Empis[3] also contends that the microscopical analysis shows that the gray granulations are entirely distinct from tubercle. On the other hand, it is alleged—and I am disposed to think with force—that the most which the investigations of M. Robin and others in accord with him show, is, that there is no special characteristic of tubercle which will enable us to declare with certainty that it is present, and that it does not possess a structure which is the same in all stages of its development. The collateral evidence goes very far to support the view that the granulations are tubercular in character.

---

[1] "A Practical Treatise on the Diseases of Children," Philadelphia, 1870, p. 452.

[2] "Die Meningitis Tuberculosa der Erwachsenen." Berlin, 1875, p. 377.

[3] "Traité de la granulie," Paris, 1865.

The question which also arises, as to whether the inflammation precedes the tubercular deposit, or *vice versa*, is generally decided in favor of the prior appearance of the tubercles. The granulations are met with in the course of the vessels of the pia mater. This membrane is always more or less inflamed, and is thickened by the infiltration of sanguineous, serous, plastic, or purulent exudations. The granular or tubercular matter is generally deposited at the base of the brain, and in this position is doubtless the cause of the derangements of motility which constitute so prominent a feature of the disease. Its ordinary seat is along the course of the middle meningeal artery and its branches. Sometimes, though rarely, it is found on the convexity of the brain.

The tissue of the brain is not generally much involved, although on section the red points, indicative of the situation of blood-vessels, are very much increased in number. Occasionally there are small extravasations of blood found in the gray substance.

The ventricles are distended by serum, and this is sometimes so great in quantity as to cause the rupture of the septum lucidum. The liquid is either clear and limpid, milky from the presence of pus-globules, or bloody from containing red corpuscles.

The morbid anatomy of the lungs and other organs, although interesting in the present connection, need not be dwelt upon; suffice it to say that tubercular deposits are always met with in some one or more of the viscera and especially in the lungs.

Treatment.—In regard to a disease so uniformly fatal as tubercular meningitis, there is not much to say. The principal advice I have to give is, to refrain from blisters, antimonial ointment, leeches, and drastic purgatives, which have no other effect than to shorten the life of the patient, and to make his existence still more intolerable than it is made by disease. Iodide of potassium does less harm, but I have never known it do any good. Niemeyer, however, recommends it, and many will doubtless continue to employ it on his authority. Seitz,[1] in a work of nearly four hundred pages, treating of tubercular meningitis in adults, devotes less than two pages to the subject of treatment, and speaks rather flippantly of all supposed remedial measures.

When we have any reason to suspect an hereditary tendency to tubercular meningitis, prophylactic measures may be used with hope of success. These consist in providing for pure air, ample clothing, nutritious food, and in the administration of cod-liver oil, iron, iodine, and quinine. A climate not subject to sudden vicissitudes, and of such a character as regards warmth and dryness that the patient can spend a great portion of the day in the open air, is also a matter of prime importance.

---

[1] *Op. et loc. cit.*

CHAPTER XI.

SUPPURATIVE ENCEPHALITIS OR CEREBRITIS.

SUPPURATIVE inflammation of the brain is a very rare affection uncomplicated with meningitis. In this latter connection it has already been sufficiently considered. In the present chapter, therefore, I shall discuss it solely as an independent lesion, and mainly in reference to the subsequent formation of abscess.

Symptoms.—The symptoms of suppurative inflammation of the brain vary according to the seat of the lesion, and are rarely of such a character as to enable us to say, with any great degree of certainty, that we have a case of uncomplicated encephalitis before us. Nevertheless, certain phenomena have been recognized, and, after death, the evidences of inflammation of the brain have been discovered. But these symptoms are, many of them, met with in other cerebral disorders, and therefore cannot be regarded as pathognomonic. It is difficult, if not impossible, to arrange them in stages; and therefore, after the prodromata, I shall consider the phenomena of acute encephalitis in accordance with their relation to the several functions of the organism liable to be affected.

The premonitory symptoms are similar to those of cerebral congestion, and doubtless depend upon a like pathological condition. Thus there are vertigo, pain in the head, noises in the ears, troubles of vision, numbness, and difficulties of speech. They never, however, last as long as they do in simple congestion.

Sometimes the first-observed symptom of approaching encephalitis is an epileptiform convulsion.

In the fully-established disease the phenomena are very decided, but at the same time have no necessary or constant relation with the pathology, as similar symptoms are met with in other very different affections.

Disorders of Sensibility.—At first, there is generally hyperæsthesia; subsequently, anæsthesia. Headache is a common symptom, as it is in so many other cerebral diseases. There is no particular location of the pain—sometimes the frontal region, at others the occipital, and again the vertical or parietal regions, being its seat. It varies, likewise, as regards intensity and form, and may consist of a feeling of fullness or constriction only. It is present from the very beginning of the disease, and usually continues through its whole course.

Pains are felt in various parts of the body, are sharp and lancinating, and often attended with cramps. Cutaneous hyperæsthesia is also occasionally met with.

In the next place, there is anæsthesia, with all its accompaniments of formication, numbness, and other abnormal sensations of the kind, mainly affecting the face and upper extremities. As to the special senses, the sight is almost always deranged. There are bright flashes of light, iridescent appearances, and photophobia, all showing increased irritability of the retina. The pupils are contracted, the conjunctivæ suffused, and the eyeballs are the seat of a dull, aching pain. Subsequently, the pupils become dilated, and vision is lost. Ophthalmoscopic examination shows, in the early stages, papillary infiltration, with retinal congestion, and later, papillary atrophy and granular degeneration, the results of optic neuritis. There is also, generally, double vision, to which allusion will be more fully made directly.

The hearing is at first very acute, and even slight noises are more or less painful. Noises in the ears, of various kinds, are present. As the disease advances, the hearing becomes much impaired, and is gradually lost, in one or both ears.

The taste and smell are rarely affected.

Disorders of Motility.—As with the sensibility, the motor organs of the body at first exhibit evidences of increased excitability. Thus, there are twitchings of the muscles, mainly of those of the face, and clonic or tonic spasms. Sometimes these last for several days. Subsultus is especially noticed in the flexor tendons of the wrist.

General convulsions may take place, with or without loss of consciousness. Frequently the action is limited to one side of the body, or implicates one side of the face, or a single limb. Strabismus occurs, and double vision is produced, at this stage, from spasms of one of the ocular muscles.

This period of muscular excitation corresponds very accurately with the stage of augmented sensibility.

It is succeeded by a period of diminished motor power. Paralysis generally begins in a distant part of the body, and slowly involves one side. Thus, there may at first be a difficulty in raising the toes, or in grasping things with the fingers; then the knee becomes weak, the flexors of the thigh follow, and the whole limb drags. If the arm be the first member affected, the difficulty advances from the fingers to the elbow, and thence to the shoulder. Sometimes the morbid action extends equally on both sides of the body, and then the gait becomes weak and shuffling. The legs are spread wide apart, so as to increase the base, and keep the centre of gravity more easily within it. The knees are bent, the pelvis is flexed on the thighs, and the whole body is inclined forward. The face rarely escapes. It may be affected on one side only, in which case there is distortion, or there may be a gradual failure of muscular power on both sides. The muscles connected with the eyes almost always suffer. Ptosis is common, and external strabismus, causing double vision, accompanies it, both being produced by the implication of the third or motor oculi nerve.

One side of the face sometimes becomes permanently contracted, and thus an appearance is produced somewhat resembling that which is caused by paralysis of the opposite side. It may be distinguished from this latter condition, however, by the fact that in it the eyelids are spasmodically closed, and the side of the face much more distorted than when there is paralysis of the opposite side. The tongue is always, in my experience, prominently affected. The first sign of diminished motility is the frequency with which it is bitten, in conversation or mastication, and sometimes it is made quite sore, on one or both sides, or at the tip, from this cause. Then the patient discovers that long-continued speaking causes a sensation of fatigue, at the root of the tongue, and that a feeling as if this organ were too large for the mouth is experienced. Then articulation becomes indistinct, the words are clipped or slurred over, so that at times it is difficult for others to understand what he says.

Disorders of Intelligence.—The first indication of mental weakness is the susceptibility experienced to the influence of emotions. The patient will thus get uncontrollable fits of laughing or crying from very slight causes, and sometimes from no apparent cause. These paroxysms are frequently of mixed character, the patient passing from laughing to crying, and *vice versa.*

The memory begins to fail at a very early period, especially as regards the names of things. The enfeeblement is by no means, however, confined to words, but facts and circumstances likewise fail to be remembered. Gradually a condition of complete dementia ensues, and finally coma, with or without previous or alternating delirium.

Disorders of the Functions of Organic Life.—There is always febrile excitement in encephalitis. At first the pulse is frequent, rising to 120, but as the disease advances it falls, till toward the close it goes below the normal standard. It is characterized, according to Barras,[1] by a characteristic tremulousness (*tremblottement*), which he compares to the unequal vibrations of a cord moderately stretched. This peculiarity he attributes to irregular arterial dilatation. According to my experience, the symptom is by no means constantly met with, and it certainly is not pathognomonic, for the same peculiarity of pulse is found in several other disorders. In a case, however, now under my care, in which there is reason to suspect encephalitis and abscess, the phenomenon is present in a marked degree, not only in the radial artery, but in the temporal and the angular, as it passes between the nose and the inner angle of the orbit.

---

[1] "Bulletin de la société médicale d'émulation," Juin et Octobre, 1823.

The respiration in the first stages is not materially deranged, but later it becomes irregular and stertorous, and finally asphyxia may take place.

The temperature of the body is elevated till the fever abates, and paralysis makes its appearance. The thermometer rarely, however, goes above 103° Fahr., and is generally a degree below this point.

The digestive organs usually show more or less evidence of derangement. Constipation is always a prominent feature, and the appetite is capricious. At times the patient refuses to eat, at others he will cram his stomach with all kinds of edibles. Deglutition is often troublesome, and occasionally dangerous, from paralysis of the pharyngeal muscles. Cases are on record in which death has occurred by the food becoming impacted in the throat, and several cases have come under my own notice, in which, from a like cause, a fatal result was barely prevented by the use of very energetic measures.

Moreover, the secretions of the mouth are almost always altered either in quantity or quality, or both, and the sensibility of the tongue and faucial mucous membrane is often impaired. Hence, the patient is not aware that he has filled his mouth, and goes on cramming it with food, which makes an alimentary mass larger than can pass through the œsophagus. This, of course, even without the pharyngeal paralysis, interferes with the act of swallowing. The fæces are sometimes passed involuntarily, but this is almost entirely a feature of the last stage. Nausea and vomiting are present more or less from the very first.

There may be either retention of urine from paralysis of the bladder, or incontinence from paralysis of the sphincter. Or both conditions may coexist, giving rise to a constant dribbling.

These symptoms may be arranged in five classes, designated by the most prominent feature of each: the *paralytic*, the *comatose*, the *epileptiform*, the *apoplectiform*, and the *maniacal*.

Complications may and often do arise. Thus there may be meningitis, temporary congestions, extravasation of blood, effusion of serum, or some intercurrent visceral affection.

The tendency of acute encephalitis is to suppuration and the consequent formation of abscess, and many of the symptoms enumerated are due to the supervention of this condition. Death ensues gradually from exhaustion or asphyxia, or may take place suddenly from the bursting of the abscess into the ventricles, or upon the surface of the brain.

Causes.—No age is exempt from the disease, although it is more common in old persons than in adults of middle age or young persons.

It is probably more frequent in males than females solely from the fact that they are more subject to the exciting causes of the disease.

Among these are the inordinate use of alcoholic liquors, venereal excesses, extreme intellectual exertion, great emotional disturbance, and exposure to extreme heat.

It may also be induced by disease of the internal ear, by erysipelas affecting the head, or by severe attacks of scarlet fever, small-pox, or other eruptive disease.

The most common cause, however, is injury of the brain.

Diagnosis.—The diagnosis of suppurative encephalitis is, in the first stages, difficult if not impossible; the symptoms being common, as I have already said, to several other disorders. From cerebral hæmorrhage the distinction can be made without difficulty, for, although encephalitis may be developed with rapidity and by an apoplectic seizure, the tendency is for the subsequent phenomena to become progressively more marked, while in hæmorrhage there is a gradual amelioration. The pulse in hæmorrhage is from the first slow and regular, unless the medulla oblongata be the seat, while in encephalitis it is rapid and irregular.

Meningitis is always associated with superficial encephalitis, and hence the symptoms bear a certain amount of resemblance to those of the affection under consideration. But the latter is, in general, characterized by the facts that the paralysis is more defined, both in intensity and location; that the delirium is less acute; that the cephalalgia is not so intense, nor the delirium so prominent or constant a phenomenon.

In epilepsy the paroxysm is the main phenomenon of the disease. When this ceases, the patient in general recovers his ordinary mental faculties, but the epileptiform seizures of suppurative encephalitis are never followed by complete intellectual restoration.

The disease with which it is most likely to be confounded is that which, from its obvious characteristics, is denominated general paralysis. I know of no diagnostic marks between the two conditions, except that general paralysis is usually of longer duration, and is ordinarily characterized by a peculiar form of mental aberration—the *délire des grandeurs* of the French.

The symptoms due to tumors are often almost identical in character with those attendant on abscess. The history of the case is our only safe guide. The fact that the brain has received an injury of some kind will indicate suppurative encephalitis as the probable difficulty. A lady is, at the moment of writing this, under my charge, who has been successively treated by several of the most skillful diagnosticians of this city, at times for abscess, and again for tumor, and I venture to say that no one, without the aid of a post-mortem examination, can say which lesion exists.

Prognosis.—Suppurative encephalitis is invariably fatal, if the disease does not terminate in resolution. As Jaccoud, however, remarks, cases of alleged cure before the stage of suppuration is reached must always have an element of uncertainty about them, and do not therefore permit us to mitigate the unfavorable character of the prognosis. Drs. Gull and Sutton,[1] while stating that there is nothing in the morbid anatomy of cerebral abscess which makes it necessarily an incurable affection, admit that practically it is irremediable. In this opinion I unhesitatingly concur.

Morbid Anatomy and Pathology.—Suppurative encephalitis is a local disease restricted in its action, and hence affecting a limited and well-defined region of the cerebral tissue. This may vary from the size of a walnut to that of the closed fist, and is ordinarily irregularly spherical in shape. Although never of a diffused character, there may be, at the same time, several centres of inflammation. The part most frequently affected is the gray matter of the cerebrum—the morbid process involving the white substance in its progress. Next, the cerebellum appears to be a favorite seat. The corpora striata, and the optic thalami, are also frequently involved.

It sometimes happens that the pus which results from the inflammatory action is not collected in a cavity, but is infiltrated into the subjacent tissue. In such cases there is no well-defined abscess, but a pulpy mass is found on examination after death, consisting of the elements of the brain-substance in a more or less disorganized condition, with those of the blood intermingled with pus—the whole of a greenish-yellow color.

Again, there may be a collection of pus, but at the same time the walls are imperfectly formed, and there is infiltration to some extent. Lastly, the puriform deposit is entirely limited by a membrane consisting of connective tissue, and forming a cyst. The cerebral substance in contact with the walls of an abscess gradually breaks down, and hence the cavity undergoes constant enlargement in all directions, but especially in the lines of least resistance. If the abscess is near the surface of the hemisphere, the tendency is to enlarge toward the external periphery; if it is situated in the central part, in the corpora striata or optic thalami, the absorption of the peripheral tissue takes place in the direction of the ventricles. In the first instance, when the rupture ensues, the pus will be extravasated into the cavity of the arachnoid; in the second, it will be poured out into the ventricular cavities. In either case, coma and death will result if the amount of pus be sufficiently large. It has happened that the pus has escaped from the cranium by the nose or ear. A lady now under my charge experienced this result several weeks since; a large quantity of purulent matter making its exit through the posterior nares. She is still alive, in full possession of her reasoning faculties, and her articulation perfect, but with the loss of sight in both eyes, paralysis of the right side of the face, the left arm, and leg, and suffering the most intense and constant pain in her head. The seat of the lesion is probably partly in the right half of the pons Varolii. The suppurative action is doubtless still going on, and I regard her death as inevitable.[1]

---

[1] "Abscess of the Brain," Reynolds's "System of Medicine," vol. ii., p. 544.

The substance of the brain in contiguity with the abscess, as already stated, undergoes disintegration. This is in the nature of softening.

CHRONIC CEREBRAL ABSCESS.

Suppurative inflammation of the brain, terminating in the formation of abscess, may be of a chronic character, the course of the disease extending over several months. This is especially apt to result from disease of the internal ear.

Cases have been reported by Abercrombie,[2] Lallemand,[3] Toynbee,[4] Ribière,[5] and others, and three have come under my own observation.

Chronic abscess may also result from injuries of the brain or skull, and from suppuration set up around a clot due to extravasation of blood.

As in the acute form of the disease, there are no very characteristic symptoms indicating the formation of abscess. Indeed, in some cases there are no symptoms at all referable to the brain for the whole period of the course of the disease, till a short time before death. A great part of a lobe may be destroyed, and even both anterior lobes almost entirely obliterated, and the patient continue to manifest his ordinary degree of intelligence.

Ribière[6] has collected a number of interesting cases, several of which almost overturn some of our most definite ideas of cerebral physiology and pathology. Thus, he cites (Observation II.) the case of a man who entered the Hôpital de la Pitié, January 27, 1866. The patient was depressed, answered questions with difficulty, and complained of a violent pain in the head. The symptoms were supposed to indicate the existence of typhoid fever. Two days subsequently a purulent discharge was noticed from the right ear, and, the pain in the head persisting, the diagnosis was changed to suppurative otitis, with probable caries of the petrous portion of the temporal bone. Leeches were applied behind the ears and purgatives administered, after which the patient felt so far well that he determined to leave the hospital. He went to work again, and, on the 12th of February, attended a ball. The following morning, pus, mixed with blood, was discharged from the right ear, and, the tendency to stupor reappearing, he again presented himself at the hospital. It was then ascertained that the flow from the ear had begun several years previously, but had ceased for the two years immediately preceding his first entrance into the hospital.

---

[1] This patient died shortly after the foregoing lines were written. She gradually passed into a state of profound coma, in which state death occurred. The pus continued to be discharged in small quantity up to the last, and microscopecal examination disclosed the existence of ganglion-cells containing granular matter, oil-globules, and other remains of broken-down nervous tissue. No post-mortem examination could be obtained.

[2] "On Chronic Inflammation of the Brain and its Membranes," *Edinburgh Medical and Surgical Journal*, vol. xvi., 1818, p. 265, *et seq.*

[3] *Op. cit.*, p. 80, *et seq.*

[4] "The Diseases of the Ear," etc., Philadelphia, 1860.

[5] "Des abcès de l'encéphale consécutifs à la carie du rocher." Thèse de Paris, 1866.

[6] *Op. cit.*

On the 14th he was in a state of not very intense stupor, since he was able to complain of the pain in the head; his pulse was 60, full and hard, and pus was passing from the right auditory canal. By the 16th of February the stupor had increased. There was no paralysis, deviation of the face, nor alterations of sensibility. The patient understood questions put to him, but answered slowly and imperfectly. The eyelids were closed, light appeared to be unpleasant, and the purulent flow still continued. He died at nine o'clock that night, without convulsions.

The post-mortem examination of the head revealed the following condition:

The external auditory canal was filled with desiccated purulent matter; there was neither abscess nor abnormal redness about the ear.

The superior longitudinal sinus was gorged with blood, the veins were black and dilated; the brain appeared congested, but a yellow tint of the right cerebral lobe was noticed. At the inferior face of this lobe, where a rupture had occurred in handling the brain, a quantity of pus estimated at one hundred grammes (about three ounces) flowed out. This was of a greenish color, and of offensive odor. The cavity left was about the size of a hen's egg, and was bounded by red, indurated, and thick walls. The pus, which during life had flowed from the auditory canal, had not come from the abscess, but from the carious petrous portion of the temporal bone.

Around the abscess the substance of the brain was yellow and softened. Three-fourths of the middle and posterior lobes were infiltrated with pus and softened in texture. The capillaries were not visible to the naked eye; the convolutions of the island of Reil were not recognizable, and the neighboring convolutions were not now distinct. The corpus striatum of the right side was healthy in its anterior fourth. In the rest of its extent it was softened. The optic thalamus was also softened, as were likewise the roots of the optic nerve. We see that, in this case, as Ribière remarks, a considerable abscess had destroyed, in great part, the corpus striatum and optic thalamus, and that, nevertheless, the patient had been able to work till within a few days of his death, and was so slightly paralyzed as to be able to attend a public ball. Aside from a certain hebetude, the intellectual faculties were not deranged.

Another patient observed by Ribière presented an entire absence of cerebral troubles, no paralysis, no contractions, no convulsions; the sensibility was intact, and the intelligence was active. Nevertheless, there was a degree of stupidity expressed in the countenance, and the expression was dull. Still there is almost always some pain in the head, which may be irregular as regards its location and character, or may be confined to one particular spot.

In one of the cases under my observation, there was very acute pain, almost constant nausea or vomiting, a strong tendency to coma, and hemiplegia of the left side, coexisting with purulent discharge from the right ear. The patient, who had a short time previous suffered an attack of scarlet fever to which the ear-trouble was due, died suddenly, comatose, but without convulsion. Examination after death showed the existence of caries of the petrous portion of the temporal bone, and an abscess containing about two ounces of pus in the middle lobe of the right hemisphere. The right corpus striatum was softened in about half of its extent.

In the other case there had been profuse discharge from the right ear for several years, unattended by any cerebral symptoms except occasional pain and headache, which were supposed by the family to be due to gastric derangement, and for which no medical advice was ever asked. One morning the patient, a young lady, twenty years of age, was suddenly roused from bed by an alarm of fire. In her hurry to dress herself, and in the confusion of the moment, she struck her head against the edge of an open door. She immediately felt a severe pain in the head and cried out, but almost instantly sank down to the floor in a stupor, from which she never emerged, death ensuing within five hours. On removing the calvarium a large extravasation of pus was discovered under the arachnoid, covering the right hemisphere, and it was ascertained that an abscess, the cavity of which was as large as a small orange, had occupied the middle lobe, and had burst through the convex superior surface by rupturing the cerebral substance. The petrous portion of the temporal bone of that side was carious, and communicated by several very small openings with the abscess.

When speaking of cerebral hæmorrhage, I have referred to another case in which there was abscess of the cerebellum, produced by injury of the skull. In this instance there were notable symptoms, vertigo, convulsions, nausea, vomiting, and violent pain in the back of the head. At first there was no paralysis, but the patient subsequently became paraplegic, and died in convulsions. Examination after death disclosed an abscess, the cavity of which comprehended nearly the whole of the left lobe of the cerebellum.

Prof. Roosa,[1] while expressing the opinion that a suppurative process of the ear is probably necessary for the production of an absce of the brain, reports a case which leads him to suspect that there m be such a thing as a chronic cerebral abscess leading to disturbing au symptoms, such as tinnitus aurium, and pain in one side of the hea without any primary aural affection. He treated a gentleman, of ab twenty-nine years of age, for some months for such symptoms as ha been indicated, and when he died a cerebral abscess was found. He could hear the watch for but three inches from the left ear, which was the affected one, and the drum membrane was sunken. Prof. Roosa supposed the case to be one of chronic proliferous inflammation of the middle ear. The patient got no relief; he became very despondent on account of his tinnitus aurium, and gave up his business and died at Sag Harbor, Long Island, of malignant pustule, about two years and a half after Prof. Roosa first saw him, and three years and a half after his first aural symptoms.

---

[1] "A Practical Treatise on Diseases of the Ear, including the Anatomy of the Organ," New York, William Wood & Co., 1873, p. 446.

Dr. George A. Sterling made a post-mortem examination, and found great injection of the pia mater over the petrous portion of the temporal bone, and an abscess about the size of a ten-cent-piece in the brain-substance. It was bounded by inflammatory adhesions, and contained about ten drops of pus. The abscess was situated on the left side, in the superior lobe, one inch from the median line, and two inches from the coronal suture. In this case there had never been a suppurative inflammation of the ear.

The fact that abscess of the brain may occur without being preceded or accompanied by suppuration of the ear is beyond doubt.

Although recovery from chronic abscess of the brain never takes place, yet life is often prolonged for several years, even when there may be marked symptoms of cerebral disorder. And when death occurs it is generally suddenly, with or without obvious exciting cause.

Treatment.—The treatment of acute suppurative encephalitis is altogether palliative. Symptoms, such as pain, vertigo, and vomiting, may be controlled to a certain extent. I have derived considerable benefit from the extract of Indian hemp, given in conjunction with the bromide of potassium. The doses of Squires's extract may range from half a grain to two grains three times a day, with from thirty to forty grains of the bromide, either of potassium or sodium. The pain and irritability of the nervous system are greatly lessened by these remedies, and thus the patient's condition rendered more tolerable.

When there is reason to suspect a syphilitic origin, mercury and iodide of potassium may be administered theoretically with some prospect of success, but practically with very little benefit. The medicines should be given in frequently-repeated doses—calomel being the preferable mercurial—so as to bring the system, as soon as possible, under their influence.

Bloodletting, local and general, blisters, tartar-emetic, and other measures calculated to depress the powers of the system, are worse than useless.

In suppurative disease of the internal ear, probably due to caries of the petrous portion of the temporal bone, preventive measures against chronic abscess may do something. Leeches applied to the mastoid process, and blisters behind the ear, are indicated, and mercury with iodide of potassium will afford a chance of a beneficial result. The solution of the bichloride of mercury with iodide of potassium in water constitutes an eligible preparation. The flow of pus should be facilitated, and the propriety of trephining the mastoid cells may be a question for consideration. The management of injuries, with a view to preventing abscess, is to be conducted upon very obvious surgical principles.

NOTE.—Under the name of CEREBRIA Dr. Charles Elam[1] has described an affection of the brain which he defines as "a spontaneous, acute general inflammation of the substance of the brain uncomplicated with meningitis." Dr. Elam has, in my opinion, adduced very strong evidence of the existence of such a disease, but I am not quite sure that the symptoms and morbid anatomy are sufficiently characteristic to warrant at present its introduction into our nosology as a pathological entity. He says:

"It is a disease which may, perhaps, occur at any period of life, although I have never seen it before eight nor after thirty-six years of age. It is certainly much more frequent between ten and thirty than at any other ages. It is uniform in its commencement as its termination. It begins with vomiting, and it ends with death. The intermediate phenomena are not very striking, and the duration is from thirty-six hours to twelve days. It differs in the most marked manner from the forms of encephalitis hitherto described, in its causation, its mode of invasion, its progress, and its morbid anatomy."

I cite the following case, which will give a good idea of the affection in question:

"H. F., a boy, aged ten, previously in good health, vomited once on the morning of June 10th. In the evening I saw him, and was informed that he was then much better. He had complained slightly of headache at the moment of vomiting, but there was little or no remains of the pain afterward. He was not in bed, and seemed very much in his usual state, except some little languor. The pulse was about seventy, regular and moderate in tone. The tongue was slightly furred, and the bowels not quite so regular as in ordinary. He denied positively and repeatedly having any pain in the head, or feeling ill in any way. I could detect no such alteration in the pupils, nor such modification in any visible or perceptible organ or function, as to lead me to suspect serious disease. My prescriptions were little more than formal directions as to diet and general management.

---

[1] "On Cerebria and other Diseases of the Brain," London, 1872, p. 32.

"For reasons unnecessary to mention, I called at the house the next day, about 11 A. M. The mother said, in answer to my inquiries, that her son must be better, he had slept so well, and was, in fact, asleep still. This at once excited my suspicions, and, going up-stairs, I found the boy pulseless, rather cold, and unable to be roused to any degree of consciousness. From this condition he never rallied, and he died the same afternoon, about thirty-two hours after the vomiting.

"*Post-mortem Examination, Thirty-five Hours after Death.*—No trace of disease in the stomach, or any of the abdominal or thoracic organs. *Head.*—The sinuses a little more full than usual, but the membrane showing no signs whatever of disease. There was no effusion, except to a very trifling amount in the lateral ventricles. The brain-substance alone showed marks of pathological change, being very closely dotted with red spots; the gray matter was darker than usual, and the white matter slightly rosy. The texture of the brain seemed to be almost normal, neither being softer nor harder than the average. There was no microscopical examination made of any part of the brain; but no doubt remained on the mind that this was a case of pure, uncomplicated, idiopathic inflammation of the brain-substance."

In another case "the whole mass of the brain was so altered in texture by inflammatory action that it could not support its own weight, nor hold together. No sooner was it removed from the head, and placed on a dish, than it gave way, falling prone together and flattening like an imperfectly-made form of jelly. The commissures were all ruptured by the weight of the hemispheres. The white matter of the brain was throughout soft, and pinkish in color. On cutting it, it smeared the knife with a streaked stain. Microscopically examined there was no pus, but an abundance of exudation corpuscles."

My reasons for somewhat doubting that these were cases of "a special cerebritis, uncomplicated, general, and idiopathic," are: That the structural changes may have begun long before they were evidenced by any notable symptoms, and hence may have existed for some time before coming under Dr. Elam's notice, and that the condition discovered after death may have resulted from occlusion of some one or more of the cerebral blood-vessels. Nevertheless I am inclined to think that Dr. Elam has made out his case; at any rate, he has made a very interesting and important contribution to cerebral pathology.

CHAPTER XII.

DIFFUSED CEREBRAL SCLEROSIS.

By diffused cerebral sclerosis is to be understood a morbid condition of some part of the brain characterized by induration and atrophy of the tissue, and not distinctly circumscribed except by the anatomical limits of the region affected.

It is not a disease which can be recognized with any great degree of certainty or even of probability during life. It is, however, a well-marked pathological condition, giving rise to very prominent symptoms. Of late years the affection has not been much noticed, except incidentally, by a few writers of special treatises—though, under the name of "induration of the brain," it received considerable attention many years ago.

The symptoms by which it is characterized are by no means peculiar to it, though, when taken collectively, they give us some reason to diagnosticate sclerosis as their cause. A number of cases have come under my observation in which the lesion was probably diffused cerebral sclerosis; but I have never had the opportunity of verifying my diagnosis by post-mortem examination. The remarks, therefore, which I shall make on the morbid anatomy will mainly be based upon the studies and observations of other writers.

Symptoms.—The symptoms of diffused cerebral sclerosis, like so many other brain-affections, are connected with the mind, with sensibility, and with the power of motion. It generally makes its appearance during infancy, and produces an arrest of development in the part of the brain affected, and consequently in certain parts of the body. The initial phenomena are those of congestion and inflammation, during the course of which epileptic convulsions frequently ensue. These may be few in number, and may cease in a few days, or they may be very frequently repeated and last for several years, or during the whole life of the patient. The mind remains undeveloped, speech, if already acquired, often becomes imperfect, and, if not yet present, may never be commenced. The limbs, usually only on one side of the body, become paralyzed, and do not grow with the same rapidity as those on the sound side. Contractions are very apt to take place, from the fact, probably, that the normal degree of antagonism between the muscles is destroyed, and that those not so much paralyzed as others draw the limbs in the direction of their action. It is quite common, therefore, in the affection under consideration, to find the fingers drawn into the palm of the hand, the wrist flexed on the forearm, the forearm on the arm, and the arm drawn backward by the action mainly of the latissimus dorsi. In the lower limbs, club-feet are produced in a similar manner.

It is not uncommon, too, to find one or more senses weak or altogether lost, and the general sensibility of the body diminished on one side.

The urine and fæces are often passed involuntarily, or else the patient, from never having acquired a sense of propriety or cleanliness, passes them whenever he chooses, at any time or place.

With this general idea of the symptoms, I proceed to refer somewhat at length to its history, in the course of which I shall quote several cases in illustration of its progress.

The first to direct specific attention to the disease under consideration was M. Pinel,[1] the younger, who, in a memoir read before the French Academy of Sciences, May 27, 1822, brought forward several cases in illustration of what he denominated "induration of the brain." I quote the first case in full as a typical example of the affection:

Beler, aged eighteen years, an idiot from birth, was admitted into the Salpêtrière Hospital, June 1, 1821. The patient was paralyzed in the left arm and leg. She could not use this arm, for the hand was strongly flexed on the forearm, and could not be extended. She walked with great difficulty, dragging the left leg. Her intellectual faculties were very much restricted; she comprehended only the questions which were addressed to her relative to her health, her intelligence not extending beyond that point. She had also great difficulty in articulating the words yes and no, which were the only words she could speak. She had no particular habit, was always calm and tranquil, and had to be anticipated in all her wants. She was subject to occasional attacks of epilepsy; but, when the paroxysms came on, she had fits almost without intermission for thirty or forty hours. They returned about every twenty-five days. On the 4th of December, 1821, the patient was taken with a series of epileptic fits, almost continual in character, which lasted during four days, the paroxysms succeeding each other with inconceivable rapidity. During these continuous convulsions the right limbs were affected with violent movements. The left limbs, which had been paralyzed for a long time, were also strongly agitated, and the general sensibility was abolished. The face was red, the eyes were twisted, the dejections were passed involuntarily, the pulse was frequent and irregular, and the respiration unequal and jerking. The patient died on the fourth day, without there having been any remission in the symptoms.

Post-mortem Examination.—"General marasmus; remarkable emaciation of the paralyzed limbs. The cranium was thick, eburnated, and very hard to break. The meninges were pale and healthy. The right lobe [hemisphere] of the brain was very much smaller than the left, it was atrophied; the convolutions were almost obliterated and very small, especially in the frontal and occipital regions. They were large and deep in the inferior part. The cortical substance was thicker than it generally is; the lateral ventricle was very small and dry. The substance of the brain, throughout the whole extent of this right lobe [hemisphere], and notably above the ventricle, was of remarkable hardness, and it was torn with difficulty by the fingers, the tissue separating in longitudinal bands which converged toward the corpus striatum.

---

[1] "Recherches d'anatomie pathologique sur l'endurcissement du système nerveux," *Journal de Physiologie de Magendie*, tome ii., 1822, p. 191, *et seq.*

"The left lobe [hemisphere] of the brain, much more developed than the right, was of the softness and consistence of the healthy brain-tissue, and this condition made the alteration in the right lobe [hemisphere] more obvious."

The rest of the description refers to other organs.

In regard to this case, M. Pinel remarks that to the pathological condition, the loss of the power of motion in the whole of one side, the almost complete annihilation of the intellectual faculties, and probably the epileptic fits, are to be ascribed. The condition—which is frequent with idiots, but of which it is often difficult to estimate all the various symptoms—is ordinarily revealed less by the paralysis of the limbs than by the distortions which it determines in the feet and the hands. Three other cases are adduced, in one of which the cerebellum was also in part indurated. M. Pinel, as the result of his observations of the morbid anatomy, states that the nervous tissue resembles a compact inorganic mass; its consistence and density are those of hard-boiled white-of-egg; the cerebral substance is atrophied; it appears entirely deprived of blood-vessels—the eye perceiving no trace of capillaries. The induration appears to affect more particularly the medullary substance than the gray substance; it was never observed in this last-named tissue.

Griesinger,[1] under the name of "diffused hypertrophy of the connective tissue of the brain," describes the affection now under consideration, and refers to an interesting case reported by Isambert,[2] in which a microscopical examination of the altered tissue was made. It occurred in an idiotic child, two years of age. The ventricular walls, the great ganglia, the pons and peduncles, were solid and hard; their tissue was elastic, like caoutchouc; the nerve-tubes in the white substance were almost completely destroyed and an amorphous granular substance occupied their place; there also existed newly-formed fibrous connective tissue. In regard to such cases, Griesinger remarks that, when we are told that a hitherto healthy and well-developed child, about the period of dentition, or during the second or third year, suddenly became feverish, was attacked with convulsions and delirium, fell into a slightly soporific state, and soon afterward apparently recovered, but with the intellectual and physical development checked, the condition may be due to one of two morbid processes: either there are slight congestion and inflammation of the membranes, or there is encephalitis, which, after passing out of the acute stage, suspends further development in the affected parts. The mind, therefore, ceases to expand; walking, if begun, is arrested; speech remains as it is, or is altogether lost; one side of the body does not grow so fast as the other; and convulsions, paralysis, and contractions, are present.

---

[1] "Die Pathologie und Therapie der psychischen Krankheiten," Zweite Auflage, 1861, p. 301 Also "New Sydenham Society Translation," p. 359.

[2] "Comptes rendus et mémoire de la Société de Biologie," tome ii., 1856, p. 9.

A case in point, referred to by Griesinger, I quote from Calmeil:[1]

"M. Alfred, born at Havre, single, aged twenty-two years, came to the Bicêtre, where he resided twenty-two months: he had been an invalid since infancy.

"Until about three years of age, he had exhibited no peculiarity as regarded intelligence—resembling other children of his years.

"At this period, however, he was attacked with measles, which was considered mild in form, and from which he had nearly recovered, when he was seized with a succession of severe eclamptic paroxysms. During twelve hours, it was impossible to rouse him from the coma, and general convulsions were present almost without interruption.

"The day after, it was perceived that he was deaf, blind, and incapable of articulating the least sound; the convulsions had ceased.

"At the end of fifteen days he recovered his hearing; after a year he could say a few words; but the retinæ continued insensible to impressions of light.

"It was now perceived that he walked with a certain degree of difficulty, and that he could hardly use the right hand. At times, also, he lost consciousness, but without falling, and it was subsequently recognized that these attacks were epileptic.

"Until the age of thirteen, the intelligence of M. Alfred underwent scarcely any development, and he remained imbecile notwithstanding all the efforts made for his improvement. He nevertheless acquired a knowledge of a certain number of words, and he could make himself understood whenever he had a want to gratify.

"At the age of nineteen he presented the symptoms of an almost complete state of idiocy. He comprehended some things, and could imperfectly articulate a few words. He was not evilly disposed, but he was incapable of attending to his person, and even of eating without assistance.

"He could take a few steps by supporting himself against the wall, on articles of furniture, or a cane, but he dragged his feet on the ground, and his right leg appeared to be weaker than the left. The right arm was contracted and almost immovable. Tactile sensibility was not affected, anywhere. He did not appear to perceive objects placed immediately before his eyes, and the pupils were dilated and insensible to the sudden accession of light. As regarded the bladder and rectum, he evacuated them without seeming to exercise the least restraint of cleanliness or propriety.

---

[1] "Traité des maladies inflammatoires du cerveau," Paris, 1859, tome ii., p. 411.

"The epileptic paroxysms occurred with long intervals between them, and presented no characteristics worthy of special mention. The complexion was pale, and the body emaciated and notably weak.

"During the month of January, 1827, there was frequent cough, combined with abundant expectoration, diarrhœa, and other symptoms of phthisis." He died in February of the same year.

Autopsy.—The whole of the right side of the body was much less developed than the left side. The right arm and leg were especially emaciated and thin. "The face was free from distortion, and the cranium, without being deformed, was small and very narrow. The greater part of the cranium was abnormally thick, and contained an excessive amount of calcareous matter.

"The dura mater was without change, and did not adhere to the osseous surfaces.

"A very considerable quantity of serum was infiltrated into the meshes of the pia mater—principally toward the middle and convex surface of the two cerebral hemispheres. The pia mater was thickened, but was not adherent to the convolutions.

"The left cerebral hemisphere was notably smaller than the right; the posterior lobe being particularly remarkable for its diminution. The convolutions were flattened, and were as thin as the blade of a knife, were resistant to the touch, and were of a clear yellow color. The middle and anterior lobes were neither of them of ordinary size.

"The posterior lobe of the right hemisphere was less developed than in a healthy brain, but the number of atrophied convolutions was small.

"On cutting into the left posterior lobe with a bistoury, its tissue was found to be white, compact, homogeneous, and very resistant. It might be said that the cerebral substance had become doughy, and that an element, foreign to its nature, gave it an excessive degree of hardness.

"On the right, the atrophied convolutions of the posterior lobes were difficult to cut; their structure was compact, but the induration of the nervous tissue did not extend deeply into the thickness of the lobe.

"In all other parts of the brain the white and the gray substance, as well on the left as on the right side, were apparently, in all respects, in a healthy condition.

"The corpora striata and the optic thalami were free from change, either as regarded their volume or their structure.

"The pons Varolii, the tubercula quadrigemina, and the peduncles of the cerebrum, and cerebellum, were in a normal state.

"The spinal cord relatively, and perhaps even absolutely, appeared to be larger than was natural.

"The optic nerves were atrophied, of a glossy white color, and very hard."

Other cases, similar in general features, are adduced by Calmeil.

In the very interesting monograph of Cotard,[1] to which reference has already been made, the relation of sclerosis to atrophy of the brain is clearly pointed out. As indicating a certain set of symptoms, in existence with a definite pathological state, I quote the following case, No. XXIX. of his series.

"C., aged fifty-eight years, an inmate of the Salpêtrière since 1828, entered the infirmary on the 25th of April, 1865, under the charge of M. Charcot.

"She gave the following information, which she said she had from her mother, and from other persons who had brought her up: At the age of eighteen months she hàd three attacks of convulsions, which left her paralyzed on her right side. She had never had convulsions since. She had already begun to walk when the seizures took place, but she did not walk again till she was three years old.

"According to the information given by the superintendent of her ward, who had known her since her entrance into the hospital, her intelligence had always been weak; she was incapable of attending to herself; she could read tolerably well, and could sign her name; she had always spoken without difficulty.

"She had been employed with coarse sewing, and had invariably been docile and attached to those who took care of her.

"Her health had always been good, though she had, when about the age of twenty-five or thirty, several attacks of hysteria. Menstruation had been regular, and had ceased when she was forty-five.

"For about a year the patient had been the subject of frequent attacks of vomiting, or of epigastric pain. At the time of her admission to the infirmary, she was very much emaciated and very cachectic.

"Her intelligence did not appear to have been recently enfeebled; she could read, sign her name, and speak without difficulty.

"Her senses seemed to be intact; sight was good in both eyes, and the pupils were equal. There was no facial paralysis, and the tongue was protruded straight.

"The right arm was emaciated, atrophied, and contracted; the forearm was pronated and semi-flexed on the arm; the hand was flexed on the forearm, and inclined toward the ulnar side; the fingers were flexed in the palm of the hand, particularly the ring and little fingers; the index-finger was semi-flexed, and the thumb was extended.

"It was possible, without very great force, to bring the several parts of the limb almost into a state of extension, but, as soon as it was left to itself, it resumed its habitual position. The patient could execute a few movements with the shoulder and the elbow, but the wrist was absolutely paralyzed, and the fingers could only be moved to a very limited extent.

---

[1] "Étude sur l'atrophie partielle du cerveau," Paris, 1868, p. 49.

"The right leg was less atrophied, and there was no other deformity than a talipes equinus. The patient walked with a cane.

"The sensibility of the right side was intact, and no very notable difference of temperature was observed between the healthy and the paralyzed sides.

"The patient died May 17th, after symptoms of acute peritonitis.

"*Autopsy.*—Cancer of the stomach, circumjacent abscess, purulent peritonitis.

"No exterior deformation of the cranium; on the left side its walls were thick, doubly and triply so at some points; the frontal sinus extended to the left of the mesial line, and communicated with a large cavity situated in the orbital arch, which was composed of two thin osseous lamellæ.

"The left middle fossa was smaller than the right, and the right cerebellar fossa was smaller than the left.

"The dura mater being incised, a large quantity of serum escaped from the left side. The left hemisphere was very small, shriveled, and in length and breadth scarcely two-thirds the corresponding dimensions of the right hemisphere. The convolutions were pressed together, were hard, and of a whitish color.

"On the external face of the middle lobe, behind the posterior marginal convolution, and on the prolongation of the fissure of Sylvius, there was a deep depression running upward and backward, and three or four centimetres in length. At the bottom of this depression the convolutions were reduced to little ridges, which were hard, and of a yellow color.

"The ventricle was considerably dilated; the corpus striatum did not appear to be perceptibly diminished in volume, but the optic thalamus was hardly one-fourth as large as that of the opposite side. There was considerable atrophy of the left crura of the fornix, and of the mammary tubercle.

"The olfactory and optic nerves of the left side were apparently healthy; the tubercular quadrigemina were not atrophied.

"The right hemisphere was healthy.

"The right hemisphere of the cerebellum and the middle cerebellar peduncle of the same side were atrophied."

Examined with the microscope, the indurated convolutions of the left hemisphere presented an enormous quantity of amyloid corpuscles and of nuclei of connective tissue:

The following cases I select from others of similar character which have occurred in my own practice:

Case I.—J. S., a boy, aged five years, was brought to me in the autumn of 1869, to be treated for epilepsy. The paroxysms occurred several times a day, and had originated when the child was two years of age, in consequence, as the mother thought, of a fall.

At that time he could say a number of words, and was rapidly learning to talk; his intelligence was good, and he had been walking for several months.

But after the first convulsion he ceased to speak and to walk, though he continued up to the time I first saw him to give his attention to very striking objects, such as noisy tops, bright-colored articles, and, above all, music and soldiers. During this period he had at least six exacerbations, characterized by pain in the head, repeated convulsions, and coma.

When he was about two years and a half old it was observed that he did not move the left arm and leg so freely as the right, and soon afterward he ceased to move them at all. The toes then began to be drawn under the sole of the foot, and the heel was raised. Then the leg became flexed on the thigh, and soon afterward the fingers of the left hand and thumb were gradually bent so as to press strongly against the palm. The wrist followed, and then the forearm. Both limbs were greatly atrophied.

When he came under my examination he was having epileptic convulsions, both of the *grand* and *petit mal*, every day. There was no deformity of the skull, though it was certainly small for his age. His mind was feeble, and he did not give attention to any remarks made to him, but bright objects at once attracted his gaze, and he made efforts to get hold of them.

I examined the fundus of the eyes with the ophthalmoscope, and discovered an anæmic condition of the retinæ and atrophy of both optic disks.

I gave it as my opinion that the child was suffering from diffused cerebral sclerosis, involving the left hemisphere; and that there was scarcely any prospect of material amelioration in his mental or physical condition.

Case II.—A female, aged eight years, entered the New York State Hospital for Diseases of the Nervous System, June, 1870, having previously been a patient at my clinic at the Bellevue Hospital Medical College. When quite an infant she had suffered from epileptiform convulsions, which had been almost immediately followed by paralysis of the right upper and lower extremities. The convulsions recurred at short intervals, and atrophy of the paralyzed limbs, with contractions of the fingers, hand, and forearm, supervened. She learned to walk, however, quite well, and also to talk without any very notable defects.

Her mind was weak, and she was extremely silly in her behavior; she had never learned to read.

Under the use of the bromide of potassium her epileptic paroxysms ceased, but the contractions and atrophy of the right arm resisted treatment by galvanism and mechanical appliances. The leg acquired much more power under the treatment than it had previously possessed.

CASE III.—W. W., a gentleman, aged forty-three, came to me, December 11, 1869, to be treated for what his physician and friends regarded as softening of the brain.

About six months previously he had experienced, on awaking in the morning, great difficulty in extending the left hand and fingers, and through the whole day there was a decided tendency manifested for the latter to close, and the hand to be flexed upon the forearm; and this gradually, day after day, became stronger, till at last neither the hand nor fingers could be extended.

Then the corresponding lower extremity became involved in a similar manner, and, about a month after noticing the first symptom, he had an epileptiform convulsion, and this was repeated twice the following day. Since then the fits have occurred at intervals of four or five days. With the contractions in the limbs of the left side, there was gradually-advancing paresis until, when he came under my observation, both arm and leg were almost completely paralyzed. Atrophy of both extremities was present to an extreme degree, and sensibility and electro-muscular contractibility were almost entirely abolished.

His mind was also notably impaired. He laughed immoderately at every question I put to him, and had a decided expression of imbecility. His speech was not affected to any remarkable degree, except as regarded extreme slowness of utterance. He had, previously to his illness, been a ready and quick speaker. My diagnosis was diffused cerebral sclerosis, and I gave an unfavorable prognosis. The treatment, which will be considered under its proper head, was, however, successful to a very considerable extent.

It will be seen, from the foregoing account of the symptoms, that diffused cerebral sclerosis is characterized mainly by weakness of intellect, paralysis, and muscular contractions.

Causes.—The predisposing causes of the affection under consideration are not thoroughly understood. The disease appears to be much more frequent in infancy, although it lasts to the period of old age, and sometimes originates at an advanced time of life.

The exciting causes are likewise imperfectly known. Injuries of the skull from falls or blows, and hæmorrhagic cysts, appear to have some influence in originating the disease, but more generally it is developed, so far as we can perceive, spontaneously.

Diagnosis.—The diagnosis of diffused cerebral sclerosis must always be more or less uncertain, for the reason that the symptoms are met with in other very different affections. In children a similar set of phenomena may be the consequence of arrest of development in the brain without any alteration of its structure recognizable by our means of observation. In the case of an idiotic child affected with convulsions, hemiplegia, and muscular contractions, I found, on post-mortem examination, the left hemisphere markedly smaller than the right, but I could detect no change of any part of its structure.

Symptoms like those met with in diffused cerebral sclerosis may result from brain-tumors of various kinds.

In adults the disease is readily discriminated from cerebral hæmorrhage and embolism by the gradual character of its advance, and by the mental symptoms being more strongly pronounced. But from softening the diagnosis cannot always be made out, and an opinion must be formed from the history and phenomena in each individual case.

From thrombosis the diagnosis is equally difficult. Perhaps the distinction may be made both as regards softening and thrombosis by the facts that, though contractions are met with in both these diseases, they are not such invariable accompaniments as they are in diffused cerebral sclerosis, and that they are never, as occasionally in the latter affection, a primary symptom.

Prognosis.—The prospect of complete recovery is very gloomy, and even amelioration has hitherto been regarded as out of the question. I am inclined, however, to think, as the result of my own experience, that the condition of patients, apparently suffering from the affection in question, may be decidedly improved by suitable medical treatment. I have several times succeeded in arresting the convulsions, strengthening the mind, increasing the strength and sensibility of the paralyzed members, and relaxing the contractions. My success has been much more decided in cases which had originated late in life—probably, for the reason mainly that the disease was seen earlier in its course.

Morbid Anatomy.—This division of the subject has already been considered incidentally, to some extent, in the remarks made under the head of symptoms, and in the detail of cases quoted.

The most obvious feature detected by ordinary observation is the increased hardness and density which the cerebral tissue has acquired. This generally occupies a considerable portion of one lobe, or may extend through the whole of it, or may even affect a whole hemisphere. It is not distinctly circumscribed, but diminishes in intensity from the centre to the periphery, and, according to Pinel, never invades the gray substance.

The increased density is attended with atrophy when the disease affects the adult, and with atrophy and arrest of development when children are its subjects.

In order to understand the essential nature of the morbid process which causes the brain to become indurated, a few words in regard to cerebral histology are necessary.

Besides the nervous tissue of the brain, there is another anatomical element present which fulfills the function of binding the cells and fibres together, and giving the whole substance its normal degree of consistence. According to Virchow,[1] this, although analogous to, is different in some respects from ordinary connective tissue. He gave to it the name of neuroglia or nerve-cement.

Diffused cerebral sclerosis consists in the hypertrophy or increased formation of this tissue, and the atrophy or disappearance of the proper nervous substance. Atrophy of the brain may, however, be due to other causes than sclerosis, as in the case reported with great minuteness by Schroeder van der Kolk,[2] and several of those cited by Lallemand,[3] Turner,[4] and other writers.

Pathology.—The symptoms which result from diffused cerebral sclerosis are those which we might expect to be the consequence of a condition which essentially consists of a disappearance of that part of the brain-tissue capable of producing or transmitting nervous force, and the substitution of another histological element which is of secondary importance. They all indicate deficient cerebral power. It is with the brain as with a muscle undergoing atrophy: less force results from its action in correspondence with the advance of the process by which the characteristic anatomical elements disappear.

Doubtless, if we had the opportunity of more thorough study of the symptoms of diffused cerebral sclerosis, and comparing them with the condition of the brain as found by post-mortem examination, we should find that they varied considerably in character, according to the part affected, and we should probably have reason to believe that the nerve-cells which had disappeared—motor, sensitive, or trophic—were in exact pathological relation with the symptoms observed. This special point has been well studied by MM. Duchenne de Boulogne and Jouffroy,[5] in a recent paper, devoted to a somewhat different disease, and to which I have recently been enabled to add a few important data.

Treatment.—This division of the subject has scarcely received any attention from authors. My experience, however, has sufficed to convince me that we can occasionally improve the condition of the patient.

If there are epileptic convulsions, they may be prevented by the administration of the bromide of potassium, in doses of at least twenty grains, three times a day, to an adult. Larger doses may be necessary. On the cessation of the convulsions, it will sometimes be found that the intelligence at once begins to be developed.

The paralysis and contractions may sometimes be lessened by the persistent use of both the induced and primary galvanic currents. The first named will often in the beginning fail to act upon the muscles, in which case the latter should be employed. This is always better for the contracted muscles than the induced current. For the relief of the paralysis it should be interrupted, for the relaxation of contractions it should be constant.

---

[1] "Cellular Pathology," Chance's translation, London, 1860, p. 277.

[2] "A Case of Atrophy of the Left Hemisphere of the Brain," etc. New Sydenham Society Translation, London, 1861.

[3] *Op. cit.*

[4] "De l'atrophie partielle ou unilatérale du cervelet," etc., Paris, 1856.

[5] "De l'atrophie aiguë et chronique des cellules nerveuses de la moelle et du bulbe rachidien," etc.: *Archives de Physiologie*, No. 4, Juillet et Août, 1870, p. 499.

As regards the central lesion, I think it may occasionally be reached, when it has not had time to become very extensive or profound. And the best and really only means I know of are, the primary galvanic current passed through the brain, and the administration of the chloride of barium.

In using the galvanic current, the electrodes—wet sponges—should be applied over the mastoid processes, and kept there for a period not exceeding three minutes. Fifteen of Smee's cells will afford a current of sufficient intensity. The application should be made about every alternate day.

The chloride of barium may be given in doses of about a grain three times a day. I usually administer it in solution, according to the following formula: ℞. Barii chloridi ʒ j, aquæ dest. ℥ j, M. ft. sol.; dose, gtt. xij three times a day.

I am unable to say that these measures have actually removed the supposed sclerosis of the brain, and caused the reformation of the atrophied cells, but I am very sure that symptoms such as are attendant upon diffused cerebral sclerosis have several times been measurably dissipated by its influence. Thus, in the third case mentioned as occurring in my practice, the mind improved, the epileptic paroxysms ceased, the contractions were relaxed, the paralysis lessened, the affected limbs increased in size, and the further progress of the disease was arrested. At the present date (December 30, 1870) the gentleman is able to take care of himself, to walk tolerably well, and to use the formerly-paralyzed arm for many purposes. In three other cases a like treatment has been productive of almost as marked a degree of benefit.

CHAPTER XIII.

MULTIPLE CEREBRAL SCLEROSIS.

In multiple cerebral sclerosis the lesion involves several parts of the same ganglion, and consists of plates or nodules of sclerosed tissue scattered throughout its substance.

It is only of late years that the affection in question has been partially recognized as a distinct pathological condition, associated with certain symptoms. These symptoms were formerly, and still are to a great extent, confounded with other groups similar in several prominent features, but different altogether in anatomical relations, normal and abnormal.

Thus, under the designation of paralysis agitans, were comprehended the phenomena due to multiple cerebral sclerosis, multiple cerebrospinal sclerosis, and muscular agitation, general or local—the result of very dissimilar lesions, or without discoverable morbid changes of any kind—the one symptom of tremor sufficing to bind them together. Even by late writers the distinction is not clearly made out.

It is, in the present state of our knowledge impossible to say in all cases what part of the intra-cranial mass is affected. Still, we are not altogether without data on this point, and an attentive consideration of the symptoms will often, at least, enable us to say what ganglion of the encephalon is the main seat of the lesion. But, mindful of the fact that this work is intended to be practical, I shall not venture to deal with pathological refinements, but will point out, with as much succinctness as possible, one form of the morbid process under notice—a form which I think I am enabled to describe, from my own observations, with considerable accuracy. That form I shall designate—

MULTIPLE SCLEROSIS MAINLY AFFECTING THE HEMISPHERES.

Symptoms.—Among the first symptoms noticed in this affection is pain, which occurs in sharp paroxysms of short duration. Sometimes the sensation is as instantaneous as an electric shock. It is rarely the case that there is any extreme constant pain experienced, though a feeling of fullness or constriction is occasionally more or less permanent.

In a few cases the first observed symptom has been an epileptic paroxysm.

It is not uncommon to meet with disorders of sensibility in other parts of the body; and these may either be anæsthetic or hyperæsthetic in character. Probably the most common is a numbness of the ends of the fingers or toes, which gives the sensation of cushions when objects are touched, and which is generally confined at first to a single upper or lower extremity. Shooting pains, something like electric shocks, are also sometimes experienced. The progress of the disease is almost invariably slow, and hence several months may elapse before any disorders of motility are experienced. These, however, are the next symptoms to make their appearance, and are generally first manifested by the occurrence of tremor or trembling.

Tremor usually, but not always, is gradual in its development, and may be restricted to narrow limits. It may at first only be felt when the patient is unusually quiet, and has not his attention engaged. Thus a gentleman told me he had, for several months, only been sensible of a vibration in his arm when he lay down at night. It was then—from the description he gave me—limited entirely to the extensor indicis of the left hand, and was, in the beginning, not strong enough to move the finger. When I first saw him, several years afterward, both arms and one leg were strongly agitated.

In another case, which I saw almost from the very beginning, the tremor was restricted to the same muscle for several months, and then gradually involved the extensors and flexors of the hand. And, in several other instances which have come under my notice, the onset was equally gentle. But, as I have said, this is not always the case. A gentleman consulted me in the summer of 1870, who, after having experienced severe darting pains in the head and through the limbs on the right side, was suddenly, while in his field overlooking some work, seized with a violent trembling of the right hand, which continued for several minutes, notwithstanding his efforts to prevent it. A few days subsequently, he had another accession of a similar kind in the same limb, and by degrees the intervals became shorter, until, in the space of a month, the tremor was constantly present except when he slept, and, when I saw him, had extended to the whole arm, and to the lower extremity of the same side.

In another case, a gentleman, much addicted to excessive mental exertion, was awakened one morning by a violent agitation in his right foot. He had been under my care several months previously for severe headache and inability to sleep, for which, believing them to result from inordinate intellectual labor, I had recommended mental rest and horseback exercise. Under the use of these measures he had apparently quite recovered, but against my advice had resumed his literary labors.

He was not very confident how long the shaking of the foot had lasted, but thought it was not more than a few seconds.

Several days afterward, while writing, his right hand began to tremble slightly. He ceased his occupation, and rubbed his hand with the other. The tremor stopped for a moment only, again began, and has scarcely ever since been absent. The whole side eventually became involved.

The tendency of the tremor is always to extend. Beginning in an extremity or a group of muscles, or only in a single muscle, it goes on attacking others, until at last all the limbs and even the head may become affected. By preference, the advance of the tremor is lateral, that is, if an arm be first invaded, the leg of the same side next suffers, then the other arm, and then the corresponding leg. Usually the head is the last part attacked; but this is not always so, as I have seen several cases in which the trembling began in it.

For a long time the tremor is to some extent under volitional control. A patient, for instance, will slap his tremulous hand on his knee and for a few seconds can manage to keep it quiet, but it soon begins to shake again, and, though perhaps a second time he may arrest its movements by a like process, the period of rest is shorter. Any change of position is calculated to quiet the tremor for a time, and thus the patient is every few minutes moving his arms or legs in the attempt to get a little respite.

It is always increased by emotional disturbance of any kind. A limb which may ordinarily be but slightly tremulous, will shake violently from the excitement or anxiety produced by making a visit to a physician. The effort to keep it quiet will also often increase the tremor.

For a very considerable period after the beginning of the disease, the shaking ceases during sleep, but eventually this state affords no respite, and the patient is thus deprived still further of his physical strength.

It is not often the case that the muscles of the face are affected very early in the disease, but they frequently become involved at a later period. In several cases I have seen a constant tremor in the upper eyelid of one or both sides, and in one instance this was the first manifestation of the disease.

In another very remarkable case the first indication of tremor was perceived in the left eyeball, which was, by clonic spasms of the internal rectus muscle, kept in a state of motion producing a kind of nystagmus. The upper lid of the same eye next became affected, and then the tremor appeared in the corresponding arm. The upper lip I have several times seen tremulous, causing thereby an indistinctness in the articulation.

I have never observed other muscles supplied by the facial, or third nerve, to be involved in the tremor.

Occasionally the lower jaw is rendered tremulous from the seat of the disease being at the origin or in the course of the fifth nerve.

The tongue is sometimes affected with tremor, generally at first on only one side, and I am inclined to think that the muscles of the pharynx and larynx do not invariably escape.

The tremor is not, as some authors have asserted, only manifested when voluntary movements are performed. This is probably the case at least in the first instance with multiple cerebro-spinal sclerosis, but it certainly is not in the purely cerebral form now under consideration. Jaccoud[1] calls attention to the error which has been committed relative to this point, and my own experience is uniformly in support of the opinion he expresses.

The next symptom of importance to make its appearance is paralysis; and, when the sclerosis is limited to the hemispheres or begins in them, it always follows the tremor. On this point I have insisted in my lectures to the class of the Bellevue Hospital Medical College, as an important indication of the fact that paralysis agitans is often a cerebral disease, and I am glad to find so exact an observer as Jaccoud[2] asserting that the paralysis is often preceded by muscular agitation or trembling.

---

[1] "Traité de pathologie interne," p. 194. [2] *Op. et loc. cit.*

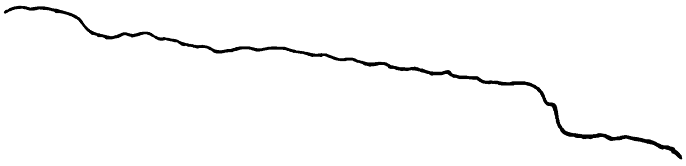
At first the loss of power is slight, and, like the trembling, is limited to a single muscle or group of muscles, but it gradually extends until it involves the limbs of one side, or even of both sides. According to my observations, it follows the course of the trembling, no limb being ever paralyzed till it has for some time been affected with tremor. In the face, however, the paralysis appears to be independent of the tremor.

The period which elapses between the appearance of the tremor and the accession of the paralysis varies in different patients, and even greatly in the same patient. Thus some muscles may exhibit notable loss of power in a few weeks after they have begun to be agitated, while others remain free from paresis for many months.

When the loss of power affects the extensors or flexors—especially in the former event—contractions may take place, as in diffused cerebral sclerosis, and the limbs are thus more or less distorted. The most common seat of this phenomenon is in the upper extremity, and it generally begins in the fingers, extending gradually to the wrist and elbow. But in some cases, even though the antagonism between certain groups of muscles be destroyed, there are no contractions. The muscles of the head, face, and trunk, do not escape. Strabismus, ptosis, and facial paralysis, are thus produced, and the muscles concerned in speech, in deglutition, and in respiration, likewise become involved. The sphincters, according to my experience, are rarely paralyzed in the early stages of the disease, but I have several times witnessed paresis of the bladder among the primary symptoms.

A marked symptom which I have observed, and which can only be distinctly shown by means of the dynamograph, is the inability of the patient to maintain a continuous muscular contraction, for even a short period. I have noticed this as among the very first indications of paresis, and I am disposed to think it exists even before the tremor is noticed. Thus, a gentleman occupying a prominent public position, and in whom I had diagnosticated multiple cerebral sclerosis mainly affecting the hemispheres, instead of making a straight line with the pencil of the instrument, traced one of which the following cut is a *fac-simile*:

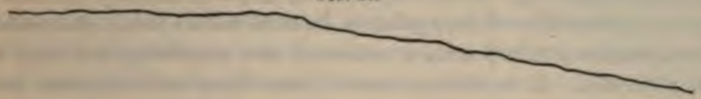
FIG. 20.



Repeated efforts only gave worse results.

In another case, that of a gentleman referred to me by my friend Dr. Van Buren, the line made was as follows:

FIG. 21.



Here the patient was able to maintain the contraction at its original force for only about the sixth of a minute—the time required for the paper to traverse the pencil being exactly half a minute, and a third part of the line being horizontal.

The ability to coördinate the affected muscles is always impaired, and thus in voluntary movements there is agitation independently of the esoteric tremor. This is seen not only in active movements, but in passive muscular contractions, such as those by which an article is held in the hand. In such a case the fingers cannot be kept in apposition with the object, but are moved about in a disorderly manner. The incoördination is manifestly connected with the inability to maintain a lengthened muscular contraction to which reference has just been made.

Sometimes, by the strong effort of the will, assisted by the sense of sight, these last two difficulties may for a little while be overcome. A gentleman now under my charge, suffering from the affection in question, cannot, for instance, carry a glass of water to his lips except by looking at it fixedly, and concentrating all his volitional power upon the act. His lower limbs are not yet affected, and he consequently can coördinate them, in walking and other movements, perfectly well.

In another case, a lady, affected with multiple cerebral sclerosis, undertook to help her invalid husband to rise from his chair; a band of music happening to pass the window, she turned to look at it, and, at once relaxing her hold, let him fall to the floor and injured him severely.

Zenker[1] reports a case in which there was a similar loss of the appreciation of the state of the muscle; and another is mentioned by Reynolds,[2] under the head of "muscular anæsthesia." I am very sure that many cases of this last-named affection are instances of multiple cerebral sclerosis of other ganglia, and I shall presently more specifically refer, under a different head, to two remarkable cases which have occurred in my own experience.

Another phenomenon closely related with this incoördination is generally present in multiple cerebral sclerosis, and that is, that the patient loses that innate or early-acquired knowledge of the exact situation of the several parts of his body. We can all of us, not thus affected, close our

[1] "Ein Beitrag zur Sklerose des Hirns und Rückenmarks," *Henle und Pfeufer's Zeitschrift für rationelle Medizin*, Bd. xxiv., 1865.

[2] "System of Medicine," vol. ii., p. 330.

eyes, and touch, with the end of the finger, any particular point on the face or rest of the body, with the utmost exactness. But a person with multiple cerebral sclerosis involving the hemispheres cannot do this. Thus, in attempting, with the eyes shut, to place the end of the index-finger on the middle of the eyebrow, he misses that point, sometimes by as much as two inches; and, no matter how frequently he tries, he succeeds no better. It would appear that, in such cases, the normal instinct of topographical relation between the fingers and the cutaneous surface generally, which all persons and many animals seem to possess, is impaired.

The electro-muscular contractility is never, according to my experience, diminished in multiple cerebral sclerosis, uncomplicated with similar lesions in the spinal cord.

The attitude and gait of a person affected with multiple cerebral sclerosis are peculiar. In standing the body is generally inclined forward, the head falling toward the chest, the trunk flexed at the pelvis, and the knees slightly bent. In walking the action is similar to a jog-trot, the body being still inclined forward, and the patient often moving with considerable rapidity. I have had several persons with the disease under my charge who could not walk at all, but who could run with surprising agility. One of these, a gentleman advanced in life, sent to me by my friend Prof. Sayre, was unable to take a step in my consulting-room. He was carried down-stairs by his attendants with some difficulty, and when he reached the front-door he was put on his feet. He then told his servant to give him a push, which the man did with all his might, and the old gentleman, being started, went at a full run and jumped into his carriage without the least difficulty.

There is often a strong tendency to plunge forward, and at times there is an impossibility of controlling it except by catching hold of some fixed object. Not long since I was walking down Broadway, when I saw in front of me a gentleman who was then under my charge, and in whom I had diagnosticated multiple cerebral sclerosis. Although aware of his peculiar impulsive gait, I had never seen it so strikingly manifested as it was then. He went at a full trot, threading his way among the numerous people in the street, until, apparently exhausted, he would lay hold of a lamp-post or awning-post and cling to it till he had recovered his breath, to start off again in a similar manner.

This impulsion of the body forward makes it easy for the patient to ascend a staircase, but, on the contrary, very difficult to go down one.

The first case of the disease in question which I saw in this city, over six years ago, was characterized by an extreme degree of festination. It was that of a maiden lady, over fifty years of age, who had been affected for several years. When she was going up-stairs no one could perceive the least irregularity in her gait, but to go down was impossible.

Sometimes, however, the tendency is to go backward. This was the case, to a remarkable extent, in a gentleman, a resident of this city, who was sent to me by Prof. Van Buren. Every time he rose from his chair he was forced to take several steps backward, and it was only by constant mental effort that he was able to go forward at all.

The tactile sensibility is generally impaired from a very early period in the course of the affection, and thus, the two points of the æsthesiometer must be more widely separated than in the normal condition of the system, in order to get two separate impressions. This anæsthesia bears no necessary relation to the region of skin covering the affected muscles. According to my experience, it is most marked at the terminal extremities of nerves.

Numbness of different degrees, pains of various kinds, increased or diminished temperature, and excessive hyperæsthesia of the skin, may also exist.

The special senses may be affected to a variable extent. Thus there may be amblyopia, or even complete blindness; the taste is very often impaired or abolished, and the hearing rendered less acute.

The ophthalmoscope should always be employed to examine the fundus of the eye. The condition generally found to exist is white atrophy of the optic disk, which is identical in general features with sclerosis. The vessels of the retina will usually be found small, the branches of the veins few in number, and the choroid of a paler hue than is natural.

The course of multiple cerebral sclerosis is progressive.

The patient is finally unable to walk, the friction of his shaking body against the bed abrades the skin, the dejections are passed involuntarily, and he dies either in coma, in convulsions, or by a gradual process of asthenia, his mind participating in the general decay. The duration of the disease varies from a few months to eight or ten years. Generally it runs its course in about five years.

Causes.—Age is certainly one of the most powerful predisposing causes of multiple cerebral sclerosis mainly affecting the hemispheres, and causing the symptoms heretofore classed as paralysis agitans. Thus, of thirteen cases in which I diagnosticated the disease in question, all were over fifty years of age, and six were over sixty. I have seen numerous cases of paralytic tremor in younger persons, but the morbid condition had scarcely any points in common with that now under notice. Cases, however, are on record in which young persons were the subjects. There is some evidence to support the theory that it is sometimes hereditary, but the whole subject is so confused in the minds of most authors, that it is difficult to make out clearly what they refer to under the designation of paralysis agitans. Of the thirteen cases occurring in my own practice, private and hospital, five had immediate ancestors who had suffered from some form of tremor and paralysis. Whether the lesion was purely cerebral, cerebro-spinal, or whether the disease was entirely functional, I was not able to decide from the information given.

The influence of sex is more readily ascertained and is very evident. Eleven of my cases were males and only two females.

Of exciting causes there are many. In two of my cases it followed immediately on attacks of scarlet fever, in two it was a sequence of typhoid fever, in two it ensued after rheumatism, in two it was probably syphilitic, in two it was apparently excited by great emotional disturbance, in one by inordinate muscular exertion, and in three no cause could be assigned, or at least there was not, in my opinion, any sufficient exciting cause to be discovered.

Diagnosis.—Multiple cerebral sclerosis has heretofore been confounded with other diseases, and its very existence as an independent affection is very illogically questioned by some writers. There is, of course, no anatomical reason why the affection should not be confined to the brain as well, as it certainly is in some cases, as to the spinal cord. To this point I will return when the morbid anatomy and pathology are discussed, and, as in the foregoing account of the symptoms and causes, will base my remarks under the present head mainly on the results of my own experience.

The occurrence of "head-symptoms" is sufficient to diagnosticate multiple cerebral sclerosis from the functional paralysis agitans, which is never a very serious affection, and the seat of which is not always centric. Besides, in the latter there are no festination, alterations of sensibility, incoördination, muscular anæsthesia, or inability to maintain a continuous muscular contraction, while the paper of the dynamograph traverses the pencil of the instrument. The functional disorder is more liable to occur in persons under fifty than in those over that age. From the cerebro-spinal form of multiple sclerosis, which will be fully considered in another section of this work, it is distinguished mainly by the facts that the tremor makes its appearance before the paralysis, and that the agitation is present whether voluntary movements are being made or not.

With the purely spinal form it is not likely to be confounded by any one paying the slightest attention to the phenomena of the two diseases.

From chorea it might in some cases not be readily discriminated without a thorough study of the clinical history and existing symptoms. But, though chorea sometimes occurs in adults, and is generally accompanied by "head-symptoms," the two affections possess few other phenomena in common.

In the first place, the mental symptoms in chorea are indicative of feebleness from the very first, while in multiple cerebral sclerosis imbecility supervenes late in the course of the disorder. In chorea there are no vertigo, pain in the head, or other evidences of congestion, while in treatment by galvanism and mechanical appliances. The leg acquired much more power under the treatment than it had previously possessed.

Case III.—W. W., a gentleman, aged forty-three, came to me, December 11, 1869, to be treated for what his physician and friends regarded as softening of the brain.

About six months previously he had experienced, on awaking in the morning, great difficulty in extending the left hand and fingers, and through the whole day there was a decided tendency manifested for the latter to close, and the hand to be flexed upon the forearm; and this gradually, day after day, became stronger, till at last neither the hand nor fingers could be extended.

Then the corresponding lower extremity became involved in a similar manner, and, about a month after noticing the first symptom, he had an epileptiform convulsion, and this was repeated twice the following day. Since then the fits have occurred at intervals of four or five days. With the contractions in the limbs of the left side, there was gradually-advancing paresis until, when he came under my observation, both arm and leg were almost completely paralyzed. Atrophy of both extremities was present to an extreme degree, and sensibility and electro-muscular contractibility were almost entirely abolished.

His mind was also notably impaired. He laughed immoderately at every question I put to him, and had a decided expression of imbecility. His speech was not affected to any remarkable degree, except as regarded extreme slowness of utterance. He had, previously to his illness, been a ready and quick speaker. My diagnosis was diffused cerebral sclerosis, and I gave an unfavorable prognosis. The treatment, which will be considered under its proper head, was, however, successful to a very considerable extent.

It will be seen, from the foregoing account of the symptoms, that diffused cerebral sclerosis is characterized mainly by weakness of intellect, paralysis, and muscular contractions.

Causes.—The predisposing causes of the affection under consideration are not thoroughly understood. The disease appears to be much more frequent in infancy, although it lasts to the period of old age, and sometimes originates at an advanced time of life.

The exciting causes are likewise imperfectly known. Injuries of the skull from falls or blows, and hæmorrhagic cysts, appear to have some influence in originating the disease, but more generally it is developed, so far as we can perceive, spontaneously.

Diagnosis.—The diagnosis of diffused cerebral sclerosis must always be more or less uncertain, for the reason that the symptoms are met with in other very different affections. In children a similar set of phenomena may be the consequence of arrest of development in the brain without any alteration of its structure recognizable by our means of observation. In the case of an idiotic child affected with convulsions, hemi-

Sometimes large numbers of amyloid corpuscles are met with, but their presence is not constant.

Pathology.—The first question to be considered under this head relates to the existence of multiple cerebral sclerosis as an independent affection—that is, without lesions of like character being at the same time produced in the spinal cord.

The weight of authority is probably against the view expressed in this chapter, and, as I have, so far as I know, made the first attempt to identify a certain group of symptoms with multiple sclerosis limited to the cerebral ganglia, I am the more desirous to place the reasons by which I have been actuated before the reader.

Andral,[1] under the designation of partial induration of the brain, describes the morbid anatomy of an affection which is probably the same as that under present consideration, although his account of it is by no means full or precise.

Valentiner,[2] citing a number of cases observed by himself and Frerichs, details one in which the lesions were limited to the brain, and in which the symptoms were similar to those I have specified in this chapter.

Jaccoud declares that certain cases establish the possibility of sclerosis limited to the encephalon. In a note he refers to several writers who have stated the parts affected, in some of which, however, the spinal cord was also involved. In the following it appears to have been restricted to the brain:

Stœhr, hemispheres corpora mamillaria; Dumville, protuberance medulla oblongata and corpora olivaria; Pool, hemispheres centrum ovale; Cruveilhier, anterior face of the medulla oblongata, protuberance, cerebral peduncles, corpus callosum, walls of the lateral ventricles, and the origins of the pneumogastric glosso-pharyngeal and hypoglossal nerves; Duplay, hemispheres, particularly in the vicinity of the ventricles, optic thalami, and corpora striata; Van Camp, protuberance; Obertimpfler, hemispheres; Barthez and Rilliet, hemispheres, particularly one convolution; Cohn, hemispheres in two cases; Gunsburg, hemisphere, gray substance of the convolutions; Valentiner-Frerichs, cerebellar peduncles, corpora olivaria, protuberance, and medulla oblongata; Meynert, cerebellum and protuberance.[3]

Bourneville and Guérard,[4] while asserting that the existence of multiple cerebral sclerosis as a separate and distinct affection rests on only one case—that of Valentiner—which they further declare was probably ence. According to Virchow,[1] this, although analogous to, is different in some respects from ordinary connective tissue. He gave to it the name of neuroglia or nerve-cement.

---

[1] "Précis d'anatomie pathologique," tome ii., 2e partie, Paris, 1829, p. 810.

[2] "Über die Sklerose des Gehirns und Rückenmarks." "Deutsche Klinik," B. xiv., 1856.

[3] I quote this note from Jaccoud, without vouching for its correctness, as, from the fact that he does not cite the works in which the details are to be found, I have not been able to verify his statements.

[4] "De la sclerose en plaques disséminées," Paris, 1869, p. 58.

Diffused cerebral sclerosis consists in the hypertrophy or increased formation of this tissue, and the atrophy or disappearance of the proper nervous substance. Atrophy of the brain may, however, be due to other causes than sclerosis, as in the case reported with great minuteness by Schroeder van der Kolk,[2] and several of those cited by Lallemand,[3] Turner,[4] and other writers.

Pathology.—The symptoms which result from diffused cerebral sclerosis are those which we might expect to be the consequence of a condition which essentially consists of a disappearance of that part of the brain-tissue capable of producing or transmitting nervous force, and the substitution of another histological element which is of secondary importance. They all indicate deficient cerebral power. It is with the brain as with a muscle undergoing atrophy: less force results from its action in correspondence with the advance of the process by which the characteristic anatomical elements disappear.

Doubtless, if we had the opportunity of more thorough study of the symptoms of diffused cerebral sclerosis, and comparing them with the condition of the brain as found by post-mortem examination, we should find that they varied considerably in character, according to the part affected, and we should probably have reason to believe that the nerve-cells which had disappeared—motor, sensitive, or trophic—were in exact pathological relation with the symptoms observed. This special point has been well studied by MM. Duchenne de Boulogne and Jouffroy,[5] in a recent paper, devoted to a somewhat different disease, and to which I have recently been enabled to add a few important data.

Treatment.—This division of the subject has scarcely received any attention from authors. My experience, however, has sufficed to convince me that we can occasionally improve the condition of the patient.

If there are epileptic convulsions, they may be prevented by the administration of the bromide of potassium, in doses of at least twenty grains, three times a day, to an adult. Larger doses may be necessary. On the cessation of the convulsions, it will sometimes be found that the intelligence at once begins to be developed.

The paralysis and contractions may sometimes be lessened by the latterly an instructor of buglers, came under my observation at Cebolleta, New Mexico, in the winter of 1849–'50. While milking a cow, one evening, he suddenly experienced a severe pain in his head, which lasted only a few seconds. He soon afterward had an epileptic paroxysm, during which he bit his tongue severely. He had no other fit, so far as was known, but the pain in the head recurred at different times, never, however, lasting longer than a minute or two.

---

[1] "Cellular Pathology," Chance's translation, London, 1860, p. 277.

[2] "A Case of Atrophy of the Left Hemisphere of the Brain," etc. New Sydenham Society Translation, London, 1861.

[3] *Op. cit.*

[4] "De l'atrophie partielle ou unilatérale du cervelet," etc., Paris, 1856.

[5] "De l'atrophie aiguë et chronique des cellules nerveuses de la moelle et du bulbe rachidien," etc.: *Archives de Physiologie*, No. 4, Juillet et Août, 1870, p. 499.

No other symptoms appeared for several weeks, and then he experienced severe darting pains in the arms, and soon afterward the left hand began to shake. On examination I found the tremor limited entirely to the extensor communis digitorum, and that the motion was entirely in the line of extension and flexion. Little by little the other muscles of the forearm became involved, and then the disorder extended upward, affecting the biceps, coraco-brachialis, triceps, deltoid, and the muscles of the shoulder generally. The arm was much weaker than the other, although he was left-handed.

In about three months after first noticing the tremor in the left hand, the left foot was similarly affected, and, as in the first instance, the agitation gradually extended upward, until, so far as I could see, all the muscles of the extremity were involved.

He now complained of numbness in the ends of the fingers of the affected extremity, and this slowly extended to the whole arm. The sensibility of the leg remained intact.

Next the right arm went through a similar sequence of phenomena, then the right leg, and finally the head.

There was no decided tendency to forward impulsion till both legs were involved, though there was difficulty in maintaining the erect posture, and the body was inclined forward before either inferior extremity became affected. But with the accession of tremor in both lower limbs, a marked disposition to trot and a corresponding difficulty of walking slowly made their appearance.

For over a year the tremor ceased whenever the patient went to sleep, and it generally became less troublesome as soon as he lay down and tried to sleep. But at last it continued night and day, and thus apparently hastened the termination of the disease, for he lost strength rapidly from deprivation of sleep. This debility was still further increased, by innutrition from proper food, it being impossible, in the then state of the country, to get any fresh vegetables.

During the whole period from the occurrence of the first paroxysm of pain, there was a gradual but marked failure of the mental powers, until a condition of very decided imbecility was reached. Death finally took place about two years and one month after the epileptic fit, which occurred on the same day with the first pain felt in the head.

I made the post-mortem examination with great care, but without any clearly-preconceived idea of what I should find, except that I expected to discover lesions of some kind in the brain and spinal cord. On removing the calvarium, the membranes covering the surface of the hemispheres were found to be healthy. I removed the entire brain from the skull, and carefully examined the base. There was no appreciable lesion of any kind. No tumor, no induration, no softening of any of the ganglia. The membranes were dissected off, and the convolutions on the superior surface were, I thought, less distinctly marked than was normal. I then cut through the right hemisphere horizontally an inch from the surface, and was surprised to find the course of the scalpel resisted by a hard body. This I discovered to be a mass of dense tissue one inch and a quarter long, half an inch wide, and about half an inch thick. I then very thoroughly examined the hemisphere, not allowing any part of it to escape observation, and discovered eleven of these nodules of variable size—the smallest as large as a cherry-stone, the largest about the size of a walnut—in the white substance. In the left hemisphere I found seven similar masses.

There were none in the peduncles, in the optic thalami, in the corpora striata, in the medulla oblongata, pons Varolii, cerebellum, or any other part of the encephalic mass.

I then examined the spinal cord in like manner, making several hundred sections of it, but found no alteration anywhere. It was perfectly healthy in every respect, neither congested, softened, nor indurated in any part of its extent.

The sclerosed bodies were, many of them, dense and as hard as cartilage, others were like hard-boiled white of egg, and others like cheese. No microscopical examination was made.

In this case the lesions were entirely limited to the hemispheres, a circumstance which I can well believe is not common—other ganglia of the brain generally participating and giving rise to corresponding modifications of, or additions to, the symptoms.

Thus, when the medulla oblongata is involved, there is difficulty of swallowing and implication of the muscles of respiration; when the pons Varolii is affected, we have among other symptoms facial paralysis; when the corpora striata, more intense paralysis; when the optic thalami, derangement of vision and perhaps of hearing; when the crura cerebri, various unilateral convulsive movements and participation of the muscles supplied by the third pair of nerves; and when the cerebellum, especially the crura, the tendency to go backward instead of forward; and so on with the other important parts of the encephalic mass.

Other relations connected with the pathology will be considered when the subjects of multiple cerebro-spinal sclerosis, and what, for want of a better name, may be called paralysis agitans, are reached.

Treatment.—To detail all the various methods which have been employed in the treatment of the group of symptoms which I have classed together as multiple cerebral sclerosis mainly affecting the hemispheres, would be a fruitless piece of labor. Many of the cases of cure which have been reported were not instances of the disease now under notice, but of the milder, and, so far as we know, functional disorder; and, therefore, it would be useless to adduce them as guides in the present connection. I shall, therefore, confine my remarks to the results of my own experience.

I am very sure that the condition of the patient is generally improved by the simultaneous administration of the chloride of barium and hyoscyamus. The former may be employed according to the formula given in the immediately preceding chapter; that is, in doses of a grain three times a day; the other in the form of the tincture, in doses of from one to two drachms morning, noon, and night. Care should be taken that the latter preparation be fresh and properly made. As sold in the apothecaries' shops, it is often inert.

By these two remedies alone, the tremor is often markedly diminished, and the paralysis and other disorders of motility and sensibility greatly lessened.

Thus, in the case of a distinguished gentleman, a Senator of the United States, who consulted me in the spring of 1870 for what was designated shaking-palsy, but in whom I diagnosticated the disease under consideration, amendment was perceived from the very first day of the treatment. The tremor and paralysis diminished, the mind became stronger and more able to endure exertion, and the physical strength much increased. He was soon able to write and to attend to his official duties, and he has continued in his advanced stage of improvement to the present date. He still, however, takes his medicines, and will probably be obliged to do so for a long time yet.

In another case—that of a gentleman living in the interior of this State—no means have been so successful in improving the general health, and arresting the progress of the disease, as the chloride of barium and tincture of hyoscyamus. I have given these remedies alone or in conjunction with others, in nine cases, and never without a decidedly favorable effect.

Electricity is, however, a powerful adjunct, and I always employ it when the opportunity exists for so doing. The primary current, from not exceeding fifteen cells, should be passed through the brain antero-posteriorly and laterally, as previously described, and the sympathetic nerve should likewise be acted upon by a current of similar intensity.

The tremulous muscles should also be subjected to the influence of a primary current of low tension. I am not sure that it makes any difference in which direction the current be passed, but it is important that it should not be so intense as to cause any considerable pain.

For the paralysis the induced current—not too strong—is to be recommended, and for any contractions that may be present it is the preferable form to use.

A gentleman, over sixty years of age, from Tennessee, consulted me in September, 1870, for tremor associated with paralysis. His physician, Dr. W. W. Yandell, came with him, and gave me much valuable information in regard to the progress of the disease. In the first place, there had been, several years previously, symptoms of a disordered cerebral circulation, indicated by pain and vertigo. Soon afterward tremor supervened in the left hand, and gradually extended to both limbs of that side. There were also paralysis and loss of sensibility. When he came under my notice, the upper extremity was more affected than the lower; contractions had taken place, and the fingers were strongly pressed against the palm of the hand, the hand was bent on the forearm, and the elbow was flexed to its utmost extent. The limb was somewhat atrophied, but electro-muscular contractility was not sensibly impaired.

The voice was exceedingly weak, but there was no paralysis of the tongue or facial muscles, and, though the patient could not speak above a whisper, every word was articulated distinctly, and was appropriately used. The body was greatly bent forward, the attitude being that of a person ascending a steep hill, and there was decided festination. The tremor and paralysis were much more marked on the left side than the right, and the agitation was altogether independent of voluntary movements.

The mind, except as regarded the memory, was not essentially impaired, and the sight and hearing were unaffected by the disease. There had never been any convulsive attack or loss of consciousness, and the course of the disease had been extremely gradual. Ophthalmoscopic examination revealed nothing beyond an anæmic condition of the retinæ and choroids.

I diagnosticated multiple cerebral sclerosis mainly affecting the hemispheres, but probably involving also the right corpus striatum, and I prescribed the chloride of barium, tincture of hyoscyamus, and electricity. He remained in New York a few days, and then returned to his home with the tremor abated, the contractions partially overcome, the muscles improved in strength, and the tendency to festination lessened.

A month afterward Dr. Yandell, who had continued the treatment, wrote me, of the patient, that the improvement was more decided than his most sanguine friends had anticipated, and still continued; that the agitation was scarcely perceptible; that he could more than half extend the fingers of the left hand, could straighten his wrist and elbow, and could lift a chair, or put on his hat, with the right hand. From what I have since ascertained, he bids fair to recover entirely.

If the general health be materially impaired, cod-liver oil, iron, and strychnia, may be administered with advantage.

The food should always be highly nutritious, and a glass or two of wine, if not particularly contraindicated, may be taken daily with advantage. Passive exercise in the open air is always beneficial, but excessive walking or strong muscular exertion of any kind should be carefully avoided. Emotional excitement or mental labor must be rigidly avoided.

Under the treatment thus indicated, the patient may at least be relieved of a great deal of his suffering.

CHAPTER XIV.

TUMORS OF THE BRAIN.

THOUGH tumors of the brain differ greatly in character, they all, when they are accompanied by any notable symptoms, present many features in common. It will, therefore, be convenient to consider them under one head, and point out their differences when the morbid anatomy and pathology are discussed.

Symptoms.—It is possible for a person to have a tumor of the brain as large as an orange, and present no symptoms of it during life. One such case came under my observation several years ago, and many others are on record. In the instance referred to, the patient, a teamster, was twice shot in a quarrel, one ball grazed the skull, ploughing up the right parietal bone for the extent of an inch; the other entered the left breast, wounding the heart. Death ensued almost instantly. The brain was examined, and a tumor of an elliptical form, two inches in its long diameter, and one and three-quarters in its short diameter, was found, involving the white substance of the left posterior lobe. The character was that which Virchow has since called gliomatous, and contained no nervous tissue.

Again, it sometimes happens that tumors of large size exist in the brain, and produce no symptoms till a few days before death. Then very violent manifestations ensue, and the patient dies convulsed or comatose. And it is always the case that the symptoms are entirely different, as one or other part of the brain is involved, or the tumor is large or small. Thus, we know very well that a morbid growth, seated in the pons Varolii, will cause very diverse symptoms from those produced by a similar formation in one of the anterior lobes of either of the hemispheres. We may say, in general terms, that tumors situated in the medulla oblongata, the pons, the optic thalami, the corpora striata, the crura cerebri, the cerebellum, and the convex surface of the hemispheres, give rise to more decided manifestations than when the white substance of the hemispheres is the seat.

Pain is probably the first symptom which attracts attention. It is generally confined to a definite region of the head corresponding to the location of the disease, but this is not always the case. It may be either a dull ache, lasting the greater portion of the day, or a sharp, lancinating paroxysm, which ensues but for a few moments and recurs frequently. As the morbid process goes on, the cephalalgia becomes more severe, and finally reaches a stage of great intensity. So great is the suffering that the patient cries out with the agony, and in a case under my observation suicide was attempted. Mental excitement, physical exertion, noises, and bright lights, aggravate the pain.

The special senses rarely escape. The sight is among the first to suffer derangement, and vision may be irretrievably lost from pressure exerted upon the optic nerve, or through congestion of the retina and choroid and consequent disorganization of these structures, or, from what is more common, optic neuritis. Indeed, it has been recently asserted by several pathologists that cerebral tumors, no matter what their situation, almost invariably give rise to this latter affection. The eyeball of the affected side is often rendered more prominent than the other, even when the tumor does not involve the orbit.

The hearing is also often affected, and the taste not unfrequently perverted or lessened in acuteness.

Disorders of sensibility in various parts of the body are common. These are either of the nature of anæsthesia or hyperæsthesia, and are usually experienced in the face or extremities.

Vertigo is a very general symptom, and may be of all degrees of intensity, sometimes preventing the patient standing, walking, or even sitting. It is often observed very early in the course of the disease, and is frequently accompanied by nausea or vomiting.

The disorders of motility are shown either as paralysis or convulsions. In several cases under my observation the loss of muscular power was first exhibited in the muscles of the eyeball and its appendages, causing external strabismus, ptosis, and permanent dilatation of the pupil, from paralysis of the third nerve, or internal strabismus from the lesion involving the sixth nerve.

In a case recently under my charge, the muscles supplied by the right facial nerve were alone affected, and in another the left side of the face and right side of the body were paralyzed. When there is paralysis, it is generally of the hemiplegic form, though occasionally it is paraplegic. Whatever its form, it is almost always of slow progress. Paralysis may be entirely absent. It is only a *necessary* attendant when the tumor involves some part of the motor tract.

When the muscles concerned in articulation are implicated, the speech is rendered indistinct, and some sounds may be impossible of utterance, not from any defect in the idea of language or of its expression, but simply from paresis of the vocal organs.

Convulsions are other prominent symptoms, and they may be among the initial phenomena. It is not at all unusual for the first evidence of intra-cranial disturbance to be an epileptiform convulsion, and similar paroxysms may occur at intervals for many years. They may be general, or, what is more common, limited to one side of the body.

Sometimes consciousness is not lost, but there are various convulsive movements of the limbs, tonic or clonic in character. Occasionally these are confined to the muscles of the face or eyeball.

Disturbances of equilibrium, manifested by tendency to advance, to go backward, or to turn round to the right or left, are sometimes present.

With these symptoms there are generally others not so palpably connected with the morbid intra-cranial process. Thus there may be disorders of the stomach, bowels, and kidneys, and of the respiration and circulation, which add much to the discomfort of the patient.

As to the intellectual faculties, it is not uncommon to find that they do not become involved to any considerable extent till a late period of the disease. Then the change is usually a gradually-advancing imbecility.

Death takes place either by convulsions or coma, or a combination of both. The following cases, which I select from my note-book, are interesting in several relations:

J. H., male, aged thirty-seven, came under my observation January 15, 1856, at Fort Riley, in Kansas. A few months before he had received an injury of the left hip by being thrown from his horse, and was stunned for a few minutes. A few days afterward, as he was lying in bed, he suddenly became vertiginous, and at the same time had noises in his ears and some pain not very definitely located. He never had vertigo again, but the pain never left him night or day for several weeks. It then suddenly ceased, and did not recur till the morning of December 31st, when a sharp twinge was experienced in the front of the head, and he immediately saw every thing double. Ptosis and dilated pupil of the left eye soon supervened, and the arm of the right side became weaker. When I saw him the grasp of his hand was very feeble, and the ocular troubles very noticeable. The pain was almost constantly present, and was of the most intense character. He said it seemed as if a red-hot iron were being thrust through his brain.

He had come several miles to see me, and went home after I had given him a palliative medicine. A few days afterward a messenger came for me in great haste, with the information that the patient was dying, and requesting my attendance. On my arrival, I found that he had been dead several hours, having had repeated severe convulsions. On post-mortem examination, a tumor, spheroidal in shape, with an average diameter of an inch and a quarter, was found occupying the middle third of the inner surface of the left middle lobe, so as to press on the left crus and third nerve.

The points of interest in this case are the sudden cessation of the pain and its recurrence simultaneously with the paralysis of the third nerve, the slight paralysis of the body, and the absence of convulsions till just before the fatal termination. The ptosis, diplopia, and dilatation of the pupil, doubtless occurred at the very instant that the tumor encroached on the crus.

The history of the following case, which I saw in September, 1864, at the request of my friend Prof. Van Buren, I take from the report of Dr. F. N. Otis,[1] under whose immediate care the patient was :

Miss E., aged twenty-six, was of healthy parentage, and, though of delicate organization, had enjoyed good health up to February, 1861, when she received a fall on the ice, striking violently upon her elbow. She was not conscious of receiving any other injury at the time. At 3 A. M. of the following day she awoke with an intense pain in the top of her head, of a throbbing, lancinating character, which continued throughout the day. By night she obtained relief. No further effect from the fall was experienced until about two weeks subsequently, when she discovered a small, firm, circumscribed swelling on the crown of the head at the point where the pain had previously been felt. This swelling, which was painless, increased gradually, until, after a year, it had attained the size of half a lemon. Soon after the appearance of the tumor, Miss E. began to suffer with severe pain, confined chiefly to the vertex, of the same character as that experienced immediately after the fall. This pain would continue almost without cessation for two or three weeks, after which for a like period she would be quite free from it.

She had also occasional attacks of numbness, preceded by great drowsiness, and a cold, creeping sensation, succeeded by total loss of the power of motion, sometimes confined to a single extremity, and at others involving the entire body. These attacks usually came on at night, or after rest in a recumbent position, and generally, though not invariably, were precursors of severe headache. They were always followed by great nervous prostration. At first rare, they increased in frequency as the tumor enlarged, so that by February, 1863, she was seldom free from them for more than ten or twelve days, and the tumor had doubled in size within the year. She now began to be much annoyed by tingling, crawling sensations in her face and through the head after any unusual exertion in writing, reading, or singing, but rode daily on horseback with apparent benefit. As time passed, she had frequent dizzy turns, with nausea, and sudden flashes like electric shocks passing over the entire body, lasting only for an instant, but leaving her much prostrated. The headache, which was always of the most agonizing description, came to be referred chiefly to the tumor, though often associated with pain through the temples and other parts of the head. The muscles of the neck sometimes became rigid, and the vision, as well as the sense of taste and smell, often became very imperfect and continued so for weeks. Sometimes the power of speech would be lost, but she always retained perfect consciousness. These attacks rarely lasted more than an hour or two.

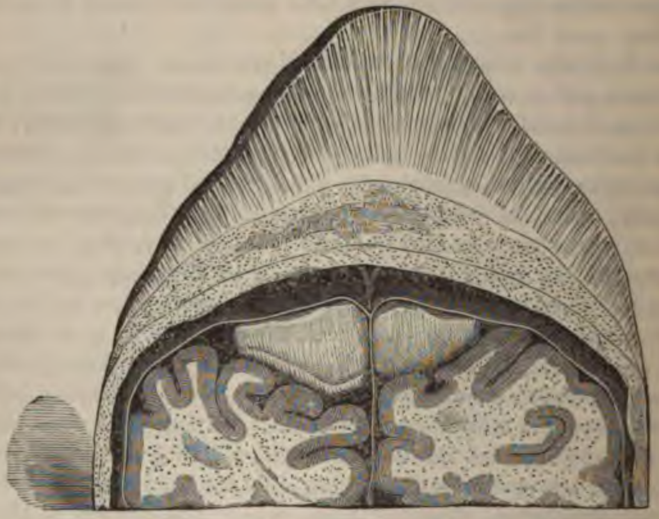
---

[1] *New York Medical Journal*, vol. i., 1865, p. 26.

On the 23d of October, 1864, she was attacked with a peritoneal inflammation, from the effects of which she died on the ninth day thereafter. Leaving out the details of the post-mortem examination of other parts of the body, we find that an incision was made across the vertex from ear to ear, and the skin dissected from the tumor, at the apex of which it was found to be firmly adherent. The calvarium was then sawn in a line one inch above the orbital margin around to the occipital protuberance; the hemispheres of the cerebrum were then sliced, and the whole raised at the same time.

On removing the two hemispheres, which were adherent above, a tumor one and a quarter inch in thickness and three inches in diameter, of a dull lemon-yellow color, a little softer than the cerebral substance, and separated into two lateral halves, was seen springing from the central surface of the dura mater. This intra-cranial tumor had insinuated itself into the sulci between the convolutions, and the dura mater could be traced between it and the bones. The situation of the tumor, and the relation to the exterior growth, are shown in the accompanying cut:

FIG. 22.



The microscopical examination by Dr. Gouley gave indications that both formations were encephaloid in character.

Similar cases to the foregoing have been reported by Mr. Paget,[1] of London, and by the late Dr. Isaacs,[1] of this city. It will be noticed that, in the case just cited, there were neither convulsions, paralysis, anæsthesia, mental derangement, nor difficulties of speech. When I saw the young lady, not long before her death, there were no symptoms present from which it could have been inferred that a tumor occupied any part of the intra-cranial cavity.

---

[1] "Surgical Pathology," London, 1853, vol. ii., p. 221.

I. R., a general officer of volunteers during the late war, consulted me in the spring of 1870, through his brother, for what was thought to be softening of the brain. The patient was stout and well made, had no difficulty of speech, no derangement of sensibility, and no paralysis of any part of the body. His senses were remarkably acute. His memory, however, was almost entirely gone, he had forgotten the names of his children, did not even know what city he was in, and could not tell me where he had been just before coming to see me. Besides this, there was absolutely nothing. His strength was enormous, and his grip one that I shall not readily forget.

His previous history was that he had served arduously through the war, and had, on being mustered out of service, resumed his business as a lumber-merchant. No syphilitic taint could be discovered. Six months before I saw him he had been suddenly seized with an epileptiform paroxysm which was followed by agonizing pain in the head. A second convulsion ensued in about a month afterward, the pain continuing to be of the utmost severity, and almost without intermission. There was a third attack, and then the pain ceased ; but the failure of memory began to be manifested from that moment, and had gradually been becoming more pronounced.

I diagnosticated a tumor involving mainly the white substance of one of the hemispheres, situated probably in the posterior lobe, and not affecting the motor tract, or the course of any of the cranial nerves. My principal reasons for not regarding the lesion as softening were the absence of paralysis or even paresis, the integrity of all the special senses, and the absolute perfection of articulation. At the same time I regarded the matter as extremely doubtful, and I cite the case here merely as one of interest in which the disease was probably a tumor. The patient died during the first week in January of the present year (1871), but I have received no details of any post-mortem examination.

In May, 1870, I was requested by Dr. Hermann Knapp to meet him in consultation in the case of a gentleman suffering from a cerebral tumor. The morbid growth apparently occupied the right anterior lobe of the brain, and involved also the temporal region of the skull on the same side. The sight of the right eye was destroyed, and that of the left so much impaired that only strong lights or shadows could be distinguished. The lymphatic glands of the neck were very much enlarged.

The pain was most acute night and day, with scarcely an intermission. The right arm was numb and paralytic, but there was no absolute paralysis anywhere except in the ocular muscles. The mind was intact, and there had never been a convulsion.

---

[1] "Transactions of the Medical Society of the State of New York," 1859.

Under the use of the iodide of potassium and the protiodide of mercury the swelling of the cranium diminished, the swollen lymphatic glands were reduced, and the pain almost entirely abolished. I saw him several times afterward, and, when I discontinued my visits, he was doing wonderfully well. Subsequently, however, there was a return of the symptoms, and death ensued.

There was no history of syphilis in this case.

The following account of a case, in which there was a tumor of the cerebellum, I have from my friend Prof. Austin Flint, M. D.:

"In June, 1842, I was present, by invitation of Dr. James P. White, of Buffalo, at the autopsy in the case of W. R., aged about forty years. I noted at that time the following brief account of the history as stated by Dr. White, the attending physician:

"The illness was dated from the preceding February (five months), but he had previously complained of pain in the head, and lassitude. In February he had had chills, which were at first attributed to malaria. Subsequently vomiting was a prominent symptom; it occurred in the morning immediately after rising from bed. Cephalalgia was a frequent, not a constant, symptom. He referred the pain especially to the occiput. In April he left Buffalo to visit friends in Rochester. He was prostrated by the journey, and, his condition now being alarming, he returned home. Notwithstanding the treatment adopted, he gradually failed, and died June 7th.

"There had never been convulsions nor paralysis.

"*Post-mortem Examination.*—The body was considerably emaciated. There was slight opacity of the arachnoid, and in some situations a small quantity of serum was effused beneath this membrane. The effusion within the ventricles was somewhat greater than usual. With these exceptions, there were no morbid appearances, except in the cerebellum. Here was a tumor of the size of an English walnut. It was of fine consistence, and supposed to be tuberculous. There was no appearance of inflammation or softening of the cerebral substance around the tumor, which was situated in the right lobe of the cerebellum.

It was ascertained in this case that the venereal appetite had been wanting for many months before death. I recollect that Dr. White informed me at the time that vertigo was a feature in this case, and that it induced unsteadiness in the voluntary movements. Dr. White has since informed me that his recollection is now distinct as regards this point."

A very important paper on intra-cranial tumors is that of my friend Prof. Roberts Bartholow, M. D.,[1] of Cincinnati. Dr. Bartholow has discussed the relations of symptoms to lesions with great perspicuity and fullness.

---

[1] "Report on Intra-cranial Tumors; their Symptomatology and Diagnosis, with Illustrated Cases," Columbus, 1869.

This able observer divides the symptoms produced by cerebral tumors into two orders:

1. Those common to morbid growths or adventitious products in general.

2. Those peculiar to tumors in special situations.

In the first order are to be placed headache, vertigo, amaurosis, convulsions, and derangement of the intellectual and reflective faculties; in the second, alterations of sensibility, disturbances of the special senses, disorders of motility, vomiting, and urinary disorders.

Causes.—The causes of cerebral tumors are so intimately connected with their character that a classification becomes at once necessary. Following Jaccoud[1] in this respect, I shall divide them into four groups: the vascular, the parasitic, the diathetic or constitutional, and the accidental. Even with this division we shall find that our knowledge of their etiology is not extensive.

Vascular tumors are aneurisms of the cerebral arteries. The term does not include the capillary aneurisms of Bouchard and Charcot, referred to under the head of cerebral hæmorrhage, but applies only to dilatations of the larger arteries. According to Gouguenheim,[2] they are more common between the ages of fifty and sixty than at other periods of life, though cases were met with under the age of puberty. Tables given by Durand[3] are to the same effect, as is likewise the experience of Lebert,[4] Gull,[5] and others. This is what might be expected from the known proclivity of the arteries to disease after the age of fifty.

Sex appears to exert but little influence, though aneurisms of the cerebral arteries seem to be somewhat more frequent with men than women.

As exciting causes, blows on the head, falls, sudden and great physical exertion, intense emotion, or mental labor, embolism, and concentric hypertrophy of the heart, are to be mentioned.

Parasitic tumors are caused by the migration of the embryos of the cysticercus and echinococcus from other parts of the body.

Diathetic tumors are either cancerous, tuberculous, or syphilitic in character. The first named are more common during the adult period of life than any other, though they are met with at all ages. Although women are more subject to some forms of cancerous tumors than men, yet in the brain they are far more common in the male sex. Of forty-eight cases studied by Lebert, cancer of the brain was primary in forty-five, that is, made its first appearance in this organ.

---

[1] *Op. cit.*, p. 247.

[2] "Des tumeurs anévrysmales des artères du cerveau." Thèse de Paris, 1866, p. 12.

[3] "Des anévrysmes du cerveau." Thèse de Paris, 1868, p. 87.

[4] "Klinische Wochenschrift," Berlin, Nos. 20 to 42, 1866.

[5] "Guy's Hospital Reports," third series, vol. v., 1859, p. 281, *et seq.*

Ogle,[1] of twenty-five cases of cerebral cancer, found that in thirteen the disease was confined to the brain, while, on the other hand, contrary to the generally received opinion, Dr. Mackenzie Bacon[2] found but ten primary cases out of seventy-three.

There is no doubt that cancer of the brain is sometimes the result of traumatic cause.

Tuberculous tumors of the brain are generally met with in young children, though they do occur, as in the case related by Dr. Flint just cited, in adults. They are almost always secondary to similar products in the lungs.

Syphilitic tumors are, of course, the result of the syphilitic infection of the system.

Accidental tumors may be caused by injuries, as was probably the case in one of the instances cited. Jaccoud, however, expresses the opinion that such an apparent relation is purely fortuitous, and that all we know of their etiology is that they are more common after the age of forty than before that period.

Diagnosis.—The diagnosis of cerebral tumors is sometimes almost self-evident, in others it is equally impossible. This difference is due, not only to the various situations they may occupy, but also to their diverse nature.

The presence of severe pain in the head for a long time is of itself some indication of the existence of a tumor if it is unaccompanied by febrile excitement. Epileptiform convulsions, occurring after the age of forty, should excite suspicion that their cause is to be found in a morbid growth of some kind. The character of the convulsive seizures will aid us in forming an opinion of their etiology. When produced by a tumor they are generally unilateral, the loss of consciousness is not so complete, and there is rarely subsequent stupor. The diagnosis from epilepsy is rendered more evident by the fact that, in tumor, the convulsions are seldom accompanied by mental weakness, and never by periods of actual unconsciousness. From softening the distinction can be made without much difficulty in the majority of cases. The acute pain, the integrity of the mind, and the absence of general paresis, will usually suffice. But sometimes the discrimination cannot be made, for there are cases of tumors in which there is very little pain, in which the mind is involved, and in which the paralysis is not very strongly marked.

The occurrence of very limited paralysis points to the existence of a tumor, rather than any other affection. A gentleman is now under my care, who, several years ago, had a cerebral hæmorrhage, from which he was rendered hemiplegic. He regained to a great extent his mental and physical powers, but a few days ago suddenly had diplopia from paralysis of the external rectus muscle of the left eye, by which internal strabismus was produced. As yet there have been no other head-symptoms except vertigo, with which he has suffered a great deal in the last two years, and which was excessive when the diplopia appeared. In other respects the health is good, and the mind gives no evidence of being affected. The paralysis of the external rectus is on the same side with the general hemiplegia.

---

[1] *British and Foreign Medico-Chirurgical Review*, July, 1865, p. 223.

[2] "On Primary Cancer of the Brain," London, 1865.

In my opinion, though I express it, of course, without positiveness, there is an aneurismal tumor pressing upon the sixth nerve after its emergence from the medulla oblongata, and probably affecting the left internal carotid artery. If this view be correct, other symptoms will certainly arise ere long. These will probably consist in the more extensive implication of cranial nerves, and in the supervention of hemiplegia.[1]

The diagnosis of the character of the tumor is of interest, and sometimes of importance with a view to the prognosis.

Aneurismal tumors are more common in persons of advanced age than in the young, they are more frequently accompanied by vertigo, and they are more generally indicated by paralysis of one or more of the cranial nerves. The mental symptoms are not often marked.

Parasitical tumors usually first manifest themselves by the occurrence of epileptiform convulsions, and the mental faculties do not long remain unaffected, for the reason that such products are more commonly seated in the gray substance of the brain than in the white tissue or the ganglia at the base. As these latter generally escape, troubles of motility are rare. Diathetic tumors are more easily recognized than any others, for the reason that we have other evidence of the existence of constitutional infection in the great majority of cases. As regards cancer, however, this aid is not generally afforded, the affection being usually primary, and not producing the ordinary indications of the cancerous cachexia. But, as in the case cited in full, and the others referred to, the existence of an external tumor is some indication, in connection with head-symptoms, that there is a corresponding growth within the cranium.

Tubercle may be suspected in cases presenting the symptoms of cerebral tumor, when there are indications of similar deposits in the lungs or other parts of the body, when the subject exhibits evidence of possessing the tuberculous diathesis, or when the history shows hereditary tendency.

In a patient presenting the symptoms of a tumor of the brain, its nature may safely be considered syphilitic if, in addition, his clinical history shows that he is tainted with syphilis, or has, at some former period, suffered from it.

---

[1] This patient was found dead in the water-closet of his residence shortly after the foregoing lines were written. There was no post-mortem examination.

In regard to accidental tumors or those of various anatomical characteristics, there is not much to be said of their diagnosis. There are no means by which one species can be distinguished from another, and no positive indications which can enable us to discriminate them from other tumors, except by the way of exclusion.

Prognosis.—Cerebral tumors almost uniformly lead to a fatal result, except they be syphilitic in character. In these latter there is a very considerable prospect of recovery if the proper medical treatment be adopted; and aneurismal tumors of the brain are occasionally spontaneously cured, and are perhaps sometimes amenable to treatment.

Morbid Anatomy and Pathology.—*Vascular Tumors.*—The most common seat of cerebral aneurisms is the basilar artery, and they are larger here than when any other vessel is affected. Gouguenheim gives the following table, based upon sixty-eight cases:

Basilar	17 cases.
Middle cerebral	14 "
Internal carotids	12 "
Anterior cerebral	8 "
Posterior communicating	5 "
Cerebellar	4 "
Anterior communicating	2 "
Posterior cerebral	3 "
Middle meningeal	2 "
Arterio-venous	2 "

Cerebral aneurisms do not differ in any essential particular from similar formations in other parts of the body. They are, however, smaller, rarely being as large as a walnut, and generally ranging in size from that of a cherry-stone to that of an almond.

Lebert ascertained that they were more frequently met with in the arteries of the left side of the brain than in those of the right. Gouguenheim confirms this observation. Thus of forty-one cases in which the side was determined, twenty-seven were on the left, and fourteen on the right. This difference is doubtless, in part at least, due to the fact that one of the causes of cerebral aneurisms, embolus, is more common on the left side than on the right, and in part to the circumstance that, the left common carotid arising directly from the arch of the aorta, the blood of that side has a greater degree of tension than the blood of the right side, and hence presses on the arterial walls with more force.

In a very interesting paper, Prof. W. R. Smith[2] calls attention to

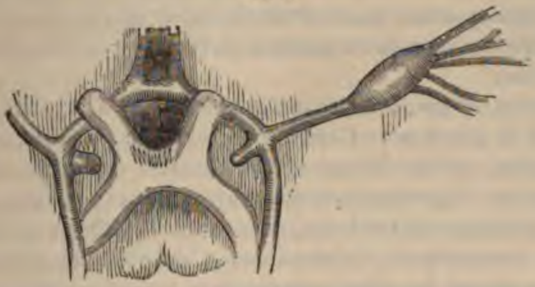
---

[1] *Op. cit.*, p. 21.

[2] "Cerebral Aneurism: Reports of the Dublin Pathological Society." *Dublin Quarterly Journal of Medical Science*, November, 1870, p. 443.

the fact that aneurisms of the encephalic arteries may be produced by embolism. The following figure, which I take from his memoir, gives an excellent illustration of such an aneurism in the left middle cerebral artery:

FIG. 28.



In regard to the post-mortem examination of the patient, from whom the preparation was taken, Prof. Smith says:

"Upon tracing the left middle cerebral artery into the fissure of Sylvius, it was found to be obstructed (just where it branches into twigs surrounding the island of Reil) by a plug of fibrine of a yellowish color and oblong form, fully a quarter of an inch in length and about the eighth of an inch in breadth. At the seat of obstruction the vessel was dilated into an oblong tumor half an inch in length and a quarter of an inch broad, the space intervening between the original plug and the arterial tunics being occupied by coagulated blood."

The theory sustained by Prof. Smith was, as he freely states, first proposed by Dr. Senhouse Kirkes[1] in the paper to which I have already referred under the head of embolism.

The idea was formerly very generally entertained, that cerebral aneurisms were always true, that is, caused by the uniform dilatation of all the coats of the artery. Hodgson[2] sustained this view on the ground that the tunics of the encephalic arteries were of such extreme tenuity that they readily dilated, and Albers,[3] Crisp,[4] Gull,[5] and others, held similar opinions, but the recognition of the fact that the arteries of the brain are peculiarly subject to disease in persons advanced in age, and the researches of Lebert, Virchow, and Kölliker, go to show that such a view is erroneous. Three other kinds are known to exist: the mixed external, in which the interior and middle coats are ruptured and the sac is formed by the external coat; the dissecting, in which the internal tunic is ruptured and the blood is to a certain extent forced between the layers of the middle tunic; and the arterio-venous. This latter is seated in the cavernous sinus, and is produced by the rupture of a small carotid aneurism, or it is the result of wound or injury.

---

[1] "Medico-Chirurgical Transactions," vol. xxxv., p. 852.

[2] "A Treatise on the Diseases of Arteries and Veins," London, 1815.

[3] "Mémoire sur les anévrysmes du cerveau et ses meninges," Bonn, 1836.

[4] "A Treatise on the Structure, Diseases, and Injuries of the Blood-vessels," London 1847.

[5] "Guy's Hospital Reports," 1857.

Aneurismal tumors may cause death either by the pressure which they exert on important parts of the brain or by the giving way of the sac and the consequent extravasation of blood, producing pressure and disorganization.

The rupture of an aneurismal tumor of course leads to the sudden development of a new set of symptoms, varying in character according to the situation of the disease and the course which the extravasated blood has taken. The extravasation may occur between the membranes, or into the substance of the brain, or into the ventricles, and is generally followed by sudden death. Occasionally, however, the patient survives to undergo a second rupture, or to die from secondary alterations of the cerebral tissue. Lebert has reported a case of aneurism of the basilar artery, in which there was a spontaneous cure; and another of the middle cerebral artery is cited by Durand[1] on the authority of Bourneville and Fremy. The process in such cases is similar to that which occurs in like cases in the extra-cranial arteries; the blood in the aneurismal sac becomes solidified, the arterial canal at this point is obliterated, and the circulation is carried on by the collateral vessels.

Parasitic tumors are of two kinds, those produced by the cysticercus and those caused by the echinococcus or hydatids. The former are small, scarcely ever being larger than a small bean. They are rarely encysted, as in other parts of the body, but are in close apposition with the brain-substance. They are generally met with in numbers ranging from ten to twenty. Cruveilhier[2] reports a case in which there were over one hundred.

They are found in all parts of the cerebrum and cerebellum; fifty of those discovered by Cruveilhier, in the case just cited, were in the cerebellum. Generally they are near the surface of the brain—often in the pia mater, in which situation they press upon the gray matter, and often in this latter substance. When situated in the ventricles, there is less impediment to the growth of the parasite, and hence it may become developed into a more or less perfect tape-worm.

Cobbold[3] states that there are about one hundred cases on record of cysticerci being found in the brain after death. Of these, Griesinger[4] reports between fifty and sixty.

Echinococci, or hydatids, though much larger than the foregoing-described parasites, are less numerous. Generally there is only one, and rarely are there two cysts. Each cyst may contain a single hydatid, as is usually the case, or there may be more in different stages of growth. In size, the cysts vary from that of a marble to that of an orange, and consist of a vascular membrane inclosing the parasite.

---

[1] *Op. cit.*, p. 14.

[2] "Anatomie pathologique générale," tome ii., p. 83, Paris, 1852.

[3] "Entozoa: An Introduction to the Study of Helminthology, with Reference more particularly to the Internal Parasites of Man," London, 1864.

[4] "Cysticerken und ihre Diagnose," "Archiv der Heilkunde," 1862.

Of one hundred and thirty-three cases occurring in the human subject and analyzed by Cobbold, sixteen were situated in the brain. All were of course fatal.

Both of these species of parasitical tumors may be primary, or they may be accompanied by similar growths in other parts of the body.

Diathetic tumors are either cancerous, tuberculous, or syphilitic.

Cancer may affect any part of the brain, though it more generally attacks the hemispheres, the cerebellum, the optic thalami, the corpus striatum, or the pons Varolii. It may begin in the bones of the cranium, in the membranes, or in the brain itself. A common seat is the orbit. According to Dr. Mackenzie Bacon,[1] of seventy-three cases of brain-tumors occurring in the London hospitals during the period from 1854 to 1863, ten were cancerous. Ladame,[2] of three hundred and thirty-nine cases of cerebral tumors, collected from various sources, found that sixty-seven were cancerous.

The dimensions of cancerous tumors are very variable. Generally they do not much exceed the size of an English walnut, though they may be twice as large.

Either variety of cancer, encephaloid, scirrhous, or colloid, may have its seat in the brain. Primitive cancer is usually single; secondary, multiple. In a case reported by Dr. Webber,[3] of Boston, in which there was a preëxisting cancerous tumor of the vagina, the brain was found to contain several deposits of cancerous growths—one quite large, situated in the left hemisphere, and two in the cerebellum.

Ogle[4] has shown that the brain-substance surrounding the cancerous growth undergoes softening. Frequently it is not changed at all.

The tumor itself does not often undergo softening, but a kind of fatty degeneration and atrophy occur, and the tissue becomes hard and compact, with no traces of blood-vessels remaining.

Tubercular tumors may be either single or multiple. In the former case, they are often as large as a cherry; in the latter, they may be as small as a grain of wheat. Very large tubercular tumors result from the fusion of two or more smaller ones. They are generally seated in the hemispheres or cerebellum, though the other parts of the encephalon are not exempt. They are the most frequently met with of all the forms of cerebral tumors.

---

[1] *Op. cit.*

[2] "Symptomatologie und Diagnostik der Hirngeschwülste," Würzburg, 1865.

[3] *Journal of Psychological Medicine*, vol. iv., 1870, p. 569.

[4] *Journal of Mental Science*, 1864, p. 229.

Syphilitic tumors or gummata are in general seated in the membranes, or in these and the gray matter at the base of the brain. They are very rarely entirely confined to the substance of the brain, and are never encysted. They are, therefore, not distinctly circumscribed, but the elements of which they are composed are infiltrated into the surrounding brain-tissue. In size they vary, rarely being as large as a walnut. Histologically they consist of nuclei and cells. The former contain nucleoli and occupy the periphery of the tumor, while the cells are found mainly in the centre. Syphilitic tumors are ordinarily accompanied by like growths in other parts of the body, especially the lungs and liver.

Accidental Tumors.—Under this head are included all formations not diathetic or vascular. Among them are the *fibro-plastic* tumors, which may attain to the size of an orange, and which are generally growths from the dura mater at the external part of the base of the cranium. They are composed of fusiform cells, nuclei, and blood-vessels. They are of variable consistence, sometimes being almost fluid, and at others gelatiniform in character.

Under the name of *glioma*, Virchow described a cerebral growth due to an abnormal development of the neuroglia or connective tissue of the brain. They are more generally found in the posterior cerebral lobes, and may attain to the size of an orange. Usually there is but one. There are two kinds of these tumors, one soft, being about the consistence of the brain-substance, the other much harder. They consist of cells and nuclei, but never contain any of the nervous elements. *Cholesteatomata*, sometimes called pearly tumors, may arise from the cranial bones, from the membranes, or from the brain itself. They rarely attain to the size of a walnut, and are generally very much smaller. Histologically they consist of a limiting membrane of extreme tenuity, the contents of which are disposed in concentric layers. These strata are epidermic cells which have undergone degeneration. There are no vessels either in the envelope or the contents, which, in addition to the elements just mentioned, consist of cholesterine and stearine.

Virchow[1] has applied the term *psammomata* to tumors composed of cerebral sand. The most common seat of these growths is the parietal dura mater at its anterior part. They are of firm consistence and are rarely larger than a cherry; microscopically they are seen to consist of isolated grains of carbonate of lime, surrounded by concentric layers of epithelium held together by connective tissue. Similar tumors are met with in the choroid plexus of the fourth verticle.

In addition to these there are *osseous tumors* (exostoses), growing from the cranial bones, and which may or may not be syphilitic, *lipomatous*, *enchondromatous*, *mucous*, *melanotic*, and several other species of tumors, which are treated of fully in the special monographs on the subject, but which need not detain us in the present connection.[1]

---

[1] "Pathologie des tumeurs," tome ii., Paris, 1869, p. 105.

Two bodies cannot occupy the same space at the same time. In a state of health, the brain so nearly fills the cranial cavity that there is barely room for those variations in the amount of blood and ventricular fluid which occur within the normal limits. The growth of a tumor, therefore, is at the expense of the brain. As the former increases in size, the latter diminishes, and hence some of the symptoms resulting from tumors are similar to those which follow atrophy or sclerosis. Besides, we have other consequent effects, such as œdema, congestion, anæmia, hæmorrhage, inflammation, or softening.

When cerebral tumors press upon the cranial nerves they produce fatty degeneration and atrophy. This effect is manifested by alterations of sensibility or of motility in the parts supplied by these nerves. In the eyes, however, in addition, the changes can be seen with the ophthalmoscope. They consist in the main of atrophy of the optic disk, disappearance of the vessels, congestion of the retina, or hæmorrhage, or serous infiltration with detachment. As Jaccoud remarks, easily appreciated by the ophthalmoscope, these lesions have a real importance in clinical diagnosis.

As to the relation between the symptoms and the seat of the lesion, the principles enunciated under the head of cerebral hæmorrhage are applicable to cerebral tumors.

Treatment.—An English surgeon, Mr. Coe,[2] reports the case of a woman, aged fifty-five, who had enjoyed good health till on one occasion she had an altercation with her husband, during which she was excited to very great anger, and in the course of which she received several severe blows on the head. About the same time she made severe efforts to lift some heavy burdens. A few minutes afterward she complained to a neighbor of a violent noise in her head—a sensation which she had never experienced before. She compared the sound to that made by the working of a fire-engine, and said that it was heard more distinctly in the left than the right ear. It was accompanied by a continuous roar similar to that of distant thunder, and this was apparently situated at the superior and posterior angle of the right parietal bone.

From the beginning of these symptoms she had not been able to lie down, but was obliged to sleep in a sitting posture. Her dreams became exceedingly frightful, and she often awoke starting and terrified.

On examination nothing abnormal could be detected in the region of the heart or great vessels, but in the neck a strong aneurismal *bruit*, synchronous with the pulse, was discovered. It was heard distinctly over the whole surface of the head, but was louder over the left temporal bone. On compressing the right common carotid artery, no effect was produced in the murmur, but pressure on the left common carotid caused it to cease at once. There was slight strabismus of the left eye, and vision was not so perfect in this eye as in the right. The hearing was not affected, but the noise in the head was so great that it overpowered the sound of the carriages in the street.

---

[1] For a very full and complete essay on the subject of Cerebral Tumors, the reader is referred to Dr. J. W. Ogle's cases illustrating the "Formation of Morbid Growths, Deposits, Tumors, Cysts, etc., in Connection with the Brain and Spinal Cord and their Investing Membranes," *British and Foreign Medico-Chirurgical Review*, 1864–'65.

[2] Cited by Gouguenheim, from *Association Medical Journal*, November, 1855.

Mr. Coe diagnosticated an aneurism of the left internal carotid artery at its entrance into the cavernous sinus immediately after its emergence from the petrous portion of the temporal bone.

On the 11th of December, 1851, Mr. Coe ligated the left common carotid artery. The *bruit* instantly ceased, but a soft and almost continuous murmur succeeded, and could be distinctly heard on applying the stethoscope to a point just above the left ear.

The patient kept the horizontal position for five hours after the operation. On the 13th there was no noise in the head, even when she concentrated her attention in the effort to hear it. From this time onward she continued to improve, and the *bruit* was never heard again.

The probability of this case being one of cerebral aneurism is of course very great, and the result leads us to believe that such tumors are not entirely beyond the reach of remedial measures. So far, however, as other tumors of the brain are concerned, there is no treatment calculated to cure the patient, unless a syphilitic taint can be ascertained to exist. It is well, however, even when there are no positive indications of the presence of such a diathesis, to act upon the presumption that it does exist, and to administer mercury in some form with the iodide of potassium. By adopting this principle, I have several times succeeded in curing patients who exhibited the most positive indications of suffering from tumor of the brain. One very remarkable case was that of a gentleman who consulted me several months since for ptosis, double vision, dilatation of the pupil, vertigo, and cephalalgia. The opinion was expressed by other physicians that there was a cerebral tumor, and I entirely accorded with the view. The gentleman had no recollection of ever having had a chancre of any kind, but I nevertheless administered the bichloride of mercury and iodide of potassium, according to the following formula: ℞. Hyd. bichlor. (corros.) gr. ij, potass. iodidi ʒ v, aquæ ℥ iv. M. ft. sol. Dose, teaspoonful three times a day. At the next visit of the patient he remembered that when in China, several years previously, he had contracted a chancre for which he was treated. I continued the treatment, conjoining it with the use of electricity to the eye so as to act upon the paralyzed muscles, and had the satisfaction to see a gradual but steady improvement take place, till eventually in the course of a few weeks the cure was complete.

Another case was that of a lady who consulted me in July, 1870, for agonizing pain in the head, vertigo, and paralysis of the third nerve of the left side, the latter producing ptosis, external strabismus, and consequent diplopia. I could discover no evidence of syphilis, but I nevertheless administered the bichloride of mercury and the iodide of potassium, as in the foregoing case. The induced or faradaic current was applied to the eye, and the patient soon began to mend. The headache disappeared first, then the vertigo, and eventually the paralysis. Subsequently I ascertained from the lady's husband that it was barely possible he might have infected his wife. I have no doubt whatever that he did.

The medication recommended can do no harm. There is, therefore, no reason why the patient should not have the chance of being benefited by it.

The prescription mentioned is a very eligible form for administering both the mercury and iodide of potassium. Salivation is never caused by it, and the stomach generally tolerates it well. Of course the proportions of the ingredients can be altered, as may seem best in individual cases.

The induced galvanic current is beneficial in restoring contractility to the paralyzed muscles. When applied to the eye the lids should be closed, one electrode, a wet sponge, is placed on them, the other is held in the hand or placed on the nape of the neck, and a current not so strong as to cause any considerable pain is then allowed to pass through the intervening tissues. For the relief of the pain attendant on cerebral tumors, morphia may be administered hypodermically, or, what I have found advantageous in several cases, the extract of Indian hemp, as recommended by Reynolds, may be used.

Counter-irritation, as produced by the actual cautery or other less powerful means, can do no possible good, and only adds to the discomfort of the patient.

CHAPTER XV.

ATHETOSIS.

UNDER the name of athetosis ('Αθετος, *without fixed position*), I propose to describe an affection which, so far as I know, had not, previous to the publication of the first edition of this work in 1871, attracted the attention of medical writers, and of which several cases have come to my knowledge. It is mainly characterized by an inability to retain the fingers and toes in any position in which they may be placed, and by their continual motion. From these phenomena, I have applied the term athetosis to the disease, having as yet had no opportunity of ascertaining by post-mortem examination the nature of the lesion to which the symptoms are due.

Since then the disease has been admitted to be well founded by several eminent pathologists, among them Dr. Clifford Allbutt,[1] Dr. Gairdner,[2] Dr. Clay Shaw,[3] Dr. C. C. Ritchie,[4] Dr. Eulenburg,[5] and Dr. Sydney Ringer.[6] It has also been studied by MM. Charcot,[7] Gairdner,[8] Oulmont,[9] Landouzy,[10] Grasset,[11] and Brousse,[12] in France, Bernhardt,[13] in Germany, and others in Europe and this country.

These symptoms will be evident from the following histories:

J. P. R., aged thirty-three, a native of Holland, consulted me September 13, 1869. His occupation was bookbinding, and he had the reputation, previous to his present illness, of being a first-class workman. He was of intemperate habits. In 1860 he had an epileptic paroxysm, and, since that time to the date of his first visit to me, had had a fit about once in every six weeks. In 1865 he had an attack of delirium tremens, and for six weeks thereafter was unconscious, being more or less delirious during the whole period.

Soon after recovering his intelligence, he noticed a slight sensation of numbness in the whole of the right upper extremity, and in the toes of the same side. At the same time severe pain appeared in these parts, and complex involuntary movements ensued in the fingers and toes of the same side.

At first the movements of the fingers were to some extent under the control of his will, especially when this was strongly exerted, and assisted by his eyesight, and he could, by placing his hand behind him, restrain them to a still greater degree. He soon, however, found that his labor was very much impeded, and he had gradually been reduced, from time to time, to work requiring less care than the finishing, at which he had been very expert.

---

[1] "Cases of Athetosis," *Medical Times and Gazette*, January 27, 1872.

[2] Cited by Dr. Clay Shaw, who gives no reference, and I have been unable to find the original.

[3] "On Athetosis; or, Imbecility with Ataxia," "St. Bartholomew's Hospital Reports," vol. ix., 1873, p. 130.

[4] "Note on a Case of Athetosis," *Medical Times and Gazette*, March 23, 1872.

[5] "Athetosis," Ziemssen's "Handbuch der speciellen Pathologie und Therapie," zwölfter Band, "Krankheiten des Nervensystems," II., zweite Hälfte, 1875, p. 389.

[6] "Notes on a Case of Athetosis, preceded by Hemiplegia and Hæmianæsthesia, and accompanied by Unilateral Sweating," *Practitioner*, August, 1877. Also, "Notes of a Post-mortem Examination in a Case of Athetosis," *Practitioner*, September, 1879.

[7] "De l'Athetose," *Leçons sur les maladies du système nerveux.* Paris, 1877.

[8] "A Case of Hammond's Athetosis," etc., *Lancet*, June 9, 1877.

[9] "Études cliniques sur l'athetose," *Thèse de Paris*, 1878.

[10] "Note sur un cas d'athetose," etc., *Progrès Médical*, 1878, Nos. 5 and 6.

[11] "Quatre nouveaux cas d'athetose," etc., Montpellier, 1879.

[12] *Montpellier Médical*, t. xxxiv., Août–Septembre, 1877.

[13] *Virchow's Archiv*, B. lxvii., H. i.

The right forearm, from the continual action of the muscles, was much larger than the other; and the muscles were hard and developed, like those of a gymnast.

When told to close his hand, he held it out at arm's length, clasped the wrist with the other hand, and then, exerting all his power, succeeded, after at least half a minute, in flexing the fingers, but instantaneously they opened again and resumed their movements.

I treated him with galvanism, primary and induced, for four months, without notable result. His fits were, however, arrested with bromide of potassium.

His memory began to be impaired soon after his attack of delirium tremens, and his intellect was manifestly weakened when I first saw him.

January 17, 1871, at my suggestion, he attended the New York State Hospital for Diseases of the Nervous System, when the following points, which I cite from the report of Dr. Cross, the Resident Physician, were noted:

The head is symmetrical, but is peculiar in shape—the posterior portion rising to a much higher point than the anterior, while the latter slopes downward and forward, giving the cranium the form of that of a Flathead Indian. The special senses are normal. The intellect is somewhat impaired, and his ideas are not so vivid at one time as at another. His memory is much enfeebled. There is slight tremor of both upper extremities, but there is no paralysis of any part of his body. There are, however, involuntary grotesque muscular movements of the fingers and toes of the right side, and these are not those of simple flexion and extension, but of more complicated form. They occur not only when he is awake, but also when he is asleep, and are only restrained by certain positions, and by extraordinary efforts of the will. Thus, those of the fingers are arrested when the wrist is firmly grasped by a strong hand, or when it is less forcibly held in a vertical position. But if the arm be extended horizontally, the fingers at once begin their movements. During their continuance the arm is hard and rigid, and the calf of the leg is also in the same state of tonic spasm while the toes are in motion. The movements are somewhat paroxysmal, being worse at times than at others. During the remissions, the power of the will over the muscles is more effective than when the paroxysms are at their height.

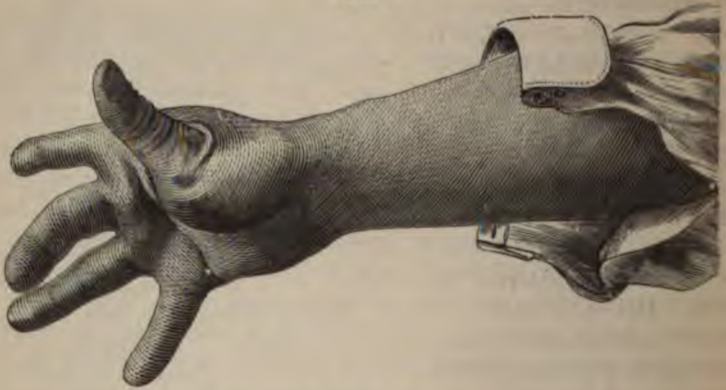
Sensibility to touch, pain, tickling, and temperature, is normal in all other parts of the body. There is slight tremulousness of the tongue, but no difficulty of articulation. There are no oscillatory movements of the eyeballs (nystagmus).

The involuntary contractions of the fingers and toes do not take place quickly, but slowly, apparently as if with deliberation, and with great force. The numbness and pain in the arm, hand, leg, and foot, have increased in proportion to the increase in the contractions.

The toes are not involved to the same degree as the fingers. Position does not, however, afford the same relief to them as to the finger and the spasms are more tonic in character. The muscular development is greater in the right arm and leg, from the almost continuou muscular action. The toes are kept restrained to some extent by th boot, but as soon as it is removed they become flexed, and take on the peculiar movements.

When, by a strong effort of the will, he succeeds for an instant i arresting the movements in the hand, the little finger at once becom strongly abducted, the third finger participates to some extent, t second finger is slightly flexed, the index-finger is extended, and t thumb is extended to its very utmost. These are the positions in cases in which he succeeds in quieting the actions, and they are w shown in the accompanying woodcut (Fig. 24), taken from a photograp h.

FIG. 24.



On account of the severe pain in the whole arm, caused by the spasms of the muscles, the patient is at times unable to go to sleep until quite exhausted. On awaking, however, after a few hours' repose, although the actions have continued during his sleep, they are not s severe as at any other time through the day or night. This state o comparative repose lasts for about half an hour.

His habits are bad. He boasts that he has often drunk as many sixty glasses of gin in a day, and it is therefore doubtful whether th tremulousness observed in the tongue and the muscles generally is th effect of the disease, or of drink, or of both combined. I have nev however, seen him drunk, or even under the influence of liquor. mental faculties are decidedly more obtuse than when he first c under my observation.

Under the use of the primary galvanic current to his brain, s cord, and affected muscles, and the internal use of chloride of ba he improved for a short time, but I have no hope of any perm result being obtained. His epileptic paroxysms are kept down with bromide of potassium.

In May, 1873, on the occasion of reading a paper on athetosis before the Medical Library and Journal Association, I brought this patient to the meeting ; and at the meeting of the American Neurological Association in this city, in June last, I again showed him as the case on which I had based my description of the disease. At that time he was in about the same condition as when he first came under my notice.[1]

Since then the patient has repeatedly come under my notice, and thus far exhibits no material change in his condition, except as regards his mental power. This is decidedly weakened. The epileptic convulsions still continue, though they are not so frequent as they once were, and are readily controlled by the bromide of potassium, or sodium, when he can be induced to take it with any approach to constancy. The muscles of the affected arm and hand are greatly hypertrophied, and he occasionally suffers from pains in both right extremities. He informs me that he has entirely stopped the use of alcoholic liquors.

The second case occurred in the practice of Dr. J. C. Hubbard, of Ashtabula, Ohio, who forwarded to me the following excellent report, dated January 11, 1870, and two photographs—one full-length on a small scale, and another, from which the woodcut, Fig. 24, has been engraved :

"H. S., aged thirty-nine years, a farmer by occupation, married. His father and paternal grandfather were free drinkers of ardent spirits. His only brother died of phthisis pulmonalis, and I think he inherits a tubercular tendency from his mother. The patient is short, muscular, is well made, and has always had good health till about eight years ago, when he had several attacks of headache, followed by vertigo and loss of power to maintain the upright posture, or to sit in a chair. After falling, he lost consciousness for a few moments. He had three of these attacks in two months.

"Three years after the last one—being five years and a half ago—while at work on a hot day in the open air, he lost consciousness and fell to the ground. This attack was more severe than the preceding ones, and he was confined to his bed three days. The headache was very severe, and continued a week after he left his bed. Aphasia and the incoördination now affecting his right forearm and right leg were the sequence of this stroke. His powers of speech were gradually reëstablished in the course of six weeks, but the impediment to normal voluntary muscular motion has remained to this day.

"In June last (1869) he applied to me for relief from cephalalgia, pain in the right side of the chest, cough, and dyspnœa. He complained also of vertigo, and of flashes of light before his eyes. His memory and judgment were slightly impaired, and he was gloomy and irritable.

---

[1] "Transactions of the American Neurological Association," vol. i., 1875, p. 17.

"His utterance of most words was perfect, but he stammered over at least one word in each sentence. It required a good deal of effort for him to connect his ideas and his sentences. He stumbled at monosyllabic words, such as *then*, *to*, *at*, *and*, and other conjunctions, but in a moment, after considerable effort, he could speak these words and conjoin his sentences correctly.

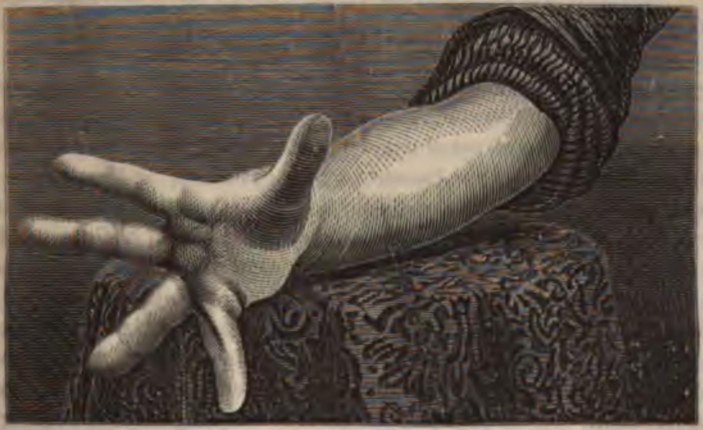
"On examining his right foot, I found that he had lost the normal antagonizing force between the flexors and extensors of the toes. The toes were ordinarily in a state of flexion, so as to present their ends to the floor. He could restore the balance in muscular action by a strong effort of the will, pressing at the same time the sole hard upon the ground, and drawing the foot backward a little. Soon, however, the extensors would be wearied by their extra work, and the toes would resume their abnormal position. The foot is slightly inverted at every step, and it is not exactly guided by the will. His gait is awkward—the foot being set down with a kind of pawing motion, as in talipes varus.

"A similar incoördination is observable in the right hand and fingers. He cannot flex his fingers without the aid of the opposite hand, but when it is closed the grasp is as strong as ever. By an intense action of the will he can keep his fist closed for a few moments, till the apparently tired flexors give way. The little and ring fingers are but partially extended, and are strongly abducted. The abductor minimi digiti and the flexor brevis minimi digiti are hypertrophied, firm, hard, and in a state of contraction most of the time, and the affected hand measures three-fourths of an inch more around the palm than its fellow. Tactile sensibility is as perfect in the affected limbs as in the others. His muscular powers are good, and he thinks he can walk twenty-five miles without injurious fatigue. The temperature of the affected limbs is slightly lower than that of the opposite ones. Has slight headache frequently, generally at evening; sleep relieves it. He sleeps well when undisturbed by pains in his limbs. Tongue clean and tremulous. Has slow-moving pains, from the hand and foot up to the body. They often last half a day, and are worse at night. Has no pain, tenderness, or feeling of weakness, in any part of the spine.

"He had no systematic treatment till last June. The chest-symptoms referred to were owing to subacute bronchitis. A seton was inserted between the shoulders, and iodide of potassium was administered for ten days. His lungs being then better, phosphoric acid, cerium, cannabis Indica, sulphate of quinine, and sulphate of iron, were given till the first of December following. He then felt so much better that he discontinued the medicines. The seton continued to discharge till the date of this communication (January 11, 1870), and he presents at this time a very marked improvement. His headache is not severe, he has less pain in his limbs, and he speaks without hesitation. By a strong effort of the will he can close his hand without assistance. He came five miles on foot, in a driving snow-storm, to see me to-day."

The accompanying woodcut (Fig. 25) is from one of Dr. Hubbard's photographs. The resemblance to the condition shown in Fig. 24 is very striking, and the histories of the two cases are so nearly identical, in regard to all essential points, as to leave no doubt that they describe instances of the same disease. Dr. Hubbard's case was, probably, when he wrote the history, in a more advanced state than is mine at the present time. The distortion of the hand is certainly greater. In the other photograph, which is indistinct, the toes are seen fully flexed.

FIG. 25.



The symptoms of athetosis are clearly indicated in the foregoing histories. Both cases came on with epileptic paroxysms—a feature accompanying other organic diseases of the brain and spinal cord. In both there are similar head-symptoms, tremulousness of the tongue, numbness on the affected side, pains in the spasmodically-affected muscles, and especially complex movements of the fingers and toes, with a tendency to distortion. In neither case is there any paralysis.

Cases of athetosis have now been reported in such numbers that further details in regard to them are scarcely necessary in this place. Eight in all have occurred in my own experience, and will be fully considered in a special monograph upon the subject. It is proper, however, in view of the fact that it is exceedingly well described, and that there was a post-mortem examination, to refer at some length to the instance, the details of which have been given by Dr. Sydney Ringer, in the two monographs already cited.

The patient, a bricklayer, woke in the morning with a severe headache, but got up and went out to walk, when a "dazzling came before his eyes," his sight became dim, he heard noises in his ears, and his face sweated profusely. These symptoms continued; he felt dizzy, and finally he fell and lost consciousness. He came to himself in a few minutes, but soon again lost consciousness. On recovering sensibility, he found that he could not speak, and was paralyzed on the right side, both as regarded motion and sensibility. All this occurred May 13, 1873.

Nearly four years afterward he was admitted into University College Hospital, and came under Dr. Ringer's observation; and it was then found that he had well-marked athetoid movements of the right upper and lower limbs. In fact, I would have no hesitation in pronouncing the case, so far as the movements are concerned, as presenting no essential difference from those which I have classed under the head of athetosis.

In regard to the nature of the disease, Dr. Ringer says: "Dazzling before the eyes, dimness of sight, giddiness preceding loss of consciousness, and followed by loss of speech and sensation, and motion of the right side, point conclusively to the left hemisphere of the cerebrum as the seat of the disease. The giddiness indicates the mesencephale, the loss of speech the posterior part of the third frontal convolution, the loss of sensation the thalamus opticus, and the loss of motion the corpus striatum, as the parts probably affected. As speech returned before sensation, and sensation before voluntary motion, the main stress of the disease must have fallen on the corpus striatum, and in a less degree on the thalamus opticus. It is probable, I think, that the cause of the disease is an embolon set free from the diseased mitral valves [the patient had an obstructive and regurgitant mitral murmur with a distinct presystolic thrill] blocking the middle cerebral artery."

The patient continued to regain his lost motor power and sensibility, and at the same time to develop increased athetoid movements. He left the hospital, but on May 29, 1878, was readmitted, and on June 12th died. Passing over those details of the post-mortem examination which relate to the heart, and which, it may be stated in general terms, was found greatly diseased, I cite from Dr. Ringer's second monograph an abstract of his very thorough report of the state of the brain:

In the left lateral ventricle there were evident signs of disease affecting the posterior part of the left corpus striatum. The left optic thalamus was decidedly smaller and flatter than the right. On section, a cyst was found occupying the posterior part of the lenticular nucleus, or, to quote Dr. Ewart's own language—the parts having been submitted to him for examination—"the cyst lies in the posterior half or two-thirds of the lenticular nucleus, the cyst lying in front immediately without the inner capsule, but passing obliquely through the lenticular nucleus. After leaving the posterior part of the lenticular nucleus it involves the whole of the white matter lying outside and rather beneath the optic thalamus, and was parallel with the left thalamus for about half an inch. A small portion of the gray matter of the optic thalamus adjacent to the cyst is destroyed."

A thorough microscopical examination of the diseased cerebral structures was made by Dr. Ewart, which is thus summed up by Dr. Ringer: "The disease in this case, therefore, appears to have been limited to the optic thalamus, corpus striatum, and the parts just external. The whole of the corpus striatum was much damaged, both the intra- and extra-lenticular portions (caudate and lenticular nuclei). Besides much atrophy and slight degeneration of the intra-venticular portion, about one-fifth of the lenticular ganglion was destroyed, and was occupied by the anterior part of the cyst. A few of the fibres (inner capsule) passing between the nuclei of the corpus striatum were destroyed. The corpus striatum was the part which suffered most; but the left optic thalamus was also wasted, and a small portion of the lower and outer portion of this body was completely destroyed, while a considerable portion of the white matter external to the thalamus (sensory fibres of the outer capsule passing from the filament) was destroyed, and the part occupied by the posterior part of the cyst. It thus appears that the diagnosis made when the man first came under my care is, in the main, correct." In my original description of athetosis I stated that, in all probability, the seat of the affection was in the gray matter of the optic thalamus or corpus striatum, and it is a gratification to find this view supported by such an array of facts as Dr. Ringer has adduced.

His case also shows that athetosis is in all probability the result of degenerative changes in the ganglionic masses mentioned, and is not produced with suddenness in any case. This is in accordance with the clinical history of all the instances which have come under my observation. Mainly in consequence of the positive data thus supplied by Dr. Ringer, I have felt justified in placing athetosis, for the present at least, in the category of diseases of the brain. And this action is still further justified by cases reported by M. Landouzy[1] and Dr. Sturges,[2] in the first of which, on post-mortem examination, there was found an old centre of softening occupying exclusively the lenticular ganglion of the corpus striatum, and entirely confined to its anterior portion. In Dr. Sturges's case it was found that, besides other changes, almost the whole of the right corpus striatum—at least that portion in front of the optic thalamus—appeared to be destroyed. In this case the paralysis had disappeared before the athetosis made its appearance.

---

[1] *Op. cit.*

[2] *Lancet*, March 15, 1879.

It would appear, therefore, that athetosis is a distinct pathological entity.

Relative to the confounding of the affection with post-hemiplegic chorea, as has been done by Charcot and others, I have only to say that the distinction between the two conditions is as well marked as between chorea and disseminated cerebro-spinal sclerosis. In athetosis the movements are slow, apparently determinate, systematic, and uniform; in post-hemiplegic chorea they are irregular, jerking, variable, and quick. Moreover, athetosis is not by any means necessarily post-hemiplegic. In the original case there had never been hemiplegia, nor was there such a state in the second case, on which my description of the disease was based. Of the eight cases which have occurred in my experience, hemiplegia was not an antecedent condition in four.

Neither is it necessarily confined to one side of the body; cases of double athetosis, without hemiplegia, having been reported by Oulmont[1] and Brousse,[2] in which there was probably general cerebral atrophy.

It is no matter for surprise that many of the cases regarded as being athetosis are not instances of that affection. This is certainly the case with many of those reported by Dr. Clay Shaw, MM. Grasset, Charcot, and others. A similar event took place when aphasia was first prominently brought to the notice of the medical profession. Every case of loss or impairment of the faculty of speech, whether from paralysis of the tongue or lips or other cause, was considered by some authors to be a case of aphasia. It was not till the disease became well known that these errors ceased to be made.

CHAPTER XVI.

MYXŒDEMA.

THE first account of the remarkable disease now known, accordi[illegible] to the suggestion of Dr. Ord,[3] as myxœdema, was given by Sir W[illegible]liam Gull.[4] He did not, however, attempt any very complete descri[illegible] tion of the cases that had come under his observation, nor enter at any length into a consideration of the morbid anatomy and pathology of the disease. His main object appeared to be to draw attention to a well-marked and probably not uncommon affection, which up to that time had not been differentiated.

---

[1] *Op. cit.*

[2] *Op. cit.*

[3] "On Myxœdema, a Term proposed to be applied to an Essential Condition in the Cr[illegible]tinoid Affection observed in Middle-aged Women."—*Medico-Chirurgical Transactions*, vol[illegible] lxi., p. 57.

[4] "On a Cretinoid State supervening in Adult Life in Women."—*Transactions of th[illegible] Clinical Society of London*, vol. vii., 1874, p. 180.

Subsequently Dr. Ord, in the paper cited, made a very thorough exposition of the symptoms and morbid anatomy of the disease, which apparently leaves little to be discovered, except so far as the minute anatomy of the nervous structures is concerned. So positive are the data furnished by the writer, that it seems to me proper to consider the affection under the head of "Diseases of the Brain."

Before the Clinical Society, October 10, 1879, Dr. Dyer Duckworth[1] reported cases of the disease, and Dr. Ord read another paper on the subject. At the same time, Dr. Sanders, of Edinburgh, in the debate which ensued, mentioned the fact that several cases which he now recognized to be instances of myxœdema, had come under his observation.

Subsequently Dr. George H. Savage[2] reported cases of this curious disorder, and gave photographs of two of Dr. Ord's cases.

In this country, the only case reported is one which occurred in my own experience, and which, with an account of what had been previously written on the disorder, formed the basis of a memoir which I read before the American Neurological Association, June 16, 1880.[3]

Since that time two cases have been reported by Dr. Thomas Inglis.[4]

This, I believe, embraces all the literature of the subject up to the present time.

Symptoms.—Myxœdema is a disease which, as Dr. Ord has shown, has for its patho-anatomical feature the deposit of a mucoid substance in various parts of the body, especially in the skin; or a degeneration and proliferation of the connective tissue. Probably both these conditions coexist in some tissues.

As a consequence of this state, an appearance resembling that of anasarca is produced, with the exception that the pressure of the finger on the part does not leave an indentation. The tissue is resilient, and not boggy like that into which water is infiltrated, as in ordinary œdema.

The face has very much the appearance, so far as the swelling is concerned, of that which is met with in cases of the toxic effect of arsenic. There is a puffiness of the eyelids, the lips are prominent, the nostrils are swollen, and the cheeks over the malar bones are red from capillary congestion.

---

[1] *Lancet*, vol. ii., 1879, p. 577.

[2] *Journal of Mental Science*, January, 1880, p. 417.

[3] "On Myxœdema, with Special Reference to its Cerebral Symptoms."—*St. Louis Clinical Record*, July, 1880, p. 97. Also, *Neurological Contributions*, No. III., 1881.

[4] *Lancet*, September 25, 1880, p. 496.

Sir William Gull was much impressed with the "spade-like" appearance, as he called it, of the hands and fingers. These latter are "clubbed," as they so frequently are in those cases of heart disease in which there is an impediment to the return of blood to the right side of the heart.

The temperature of the body is, in all cases, below the normal standard.

Thus far all the instances of the affection reported have been in adult women, unless an exception exists as regards one in a man occurring in Dr. Savage's experience, in relation to which there is some doubt as to its identity with myxœdema.

The cerebral and nervous symptoms appear to be very decided. The intellect is notably weakened, and replies to questions are given in a sluggish and inexact way. The memory is imperfect, and the patient experiences a lack of confidence in herself both as regards mental and physical power. The special senses are more or less perverted, and there are sometimes hallucinations or delusions. One case cited by Dr. Savage "was distinctly maniacal, sleepless, incoherent, violent at night." The most ordinary mental condition met with is, however, a lassitude or stupidity resembling the state generally known as acute dementia.

Such are the most marked features of the disease as described by the authorities I have mentioned.

Since the appearance of Sir William Gull's and Dr. Ord's papers, my attention has been directed to the subject, and I have been on the look-out for cases similar to those described by these gentlemen. Two instances only of the affection, but these of a most undoubted character, have as yet come under my observation:

Mrs. H. S., aged forty-one, consulted me first, April 22, 1880. I saw her again April 29th, and again May 6th. Her appearance was that of a person suffering from general œdema, the consequence of heart or kidney disease. The lower eyelids and the face immediately below them were turgid; the skin over the forehead was rough and swollen in spots; the nose was thick; the lips, especially the lower one, protruded like those of a person who has received a severe blow upon the mouth; and the skin over the malar bones was not only thickened, but for a space on each side the size of a dollar was red with a hectoid flush.

The neck was also greatly swollen, as were likewise the hands. All the fingers were "clubbed," but there was no incurvation of the nails.

Extending my inspections, I found that the whole surface of the body was similarly affected. At no place, however, could pitting be produced by pressure. As soon as the end of the finger was removed, the depressed surface returned to its ordinary level.

It was very evident that this was a case of myxœdema, and the continuance of my inquiries served to confirm the impression derived from a simple inspection of the more obvious characteristics of the case.

The general sensibility of the skin was markedly diminished. Thus, on the cheek, the two points of the æsthesiometer could barely be distinguished when separated to the extent of an inch and a half—three times more than the normal distance; and at the ends of the fingers, where they should have both been felt at a distance apart of the twelfth of an inch, they had to be separated five twelfths of an inch before each was perceived. A like condition existed in the skin of the trunk and lower extremities.

At an early period she had suffered from pains in various regions of the head, but latterly these had disappeared, and there had been no similar disturbances of sensibility in other parts of the body. On the contrary, as the æsthesiometer indicated, sensibility was diminished. The ends of the fingers felt as if there were "tight thimbles on them," to use her own expression, and the soles of her feet as though they were padded or cushioned. The various sensations of numbness were present more or less in the face, the end of the tongue, and the arms and legs.

The muscular power of the patient appeared to be decidedly weaker than was normal. The gait was staggering, the feet were not lifted clear of the ground, the grasp of the hands was feeble, and the articulation was sluggish and indistinct. There was marked difficulty of co-ordination both in the upper and lower extremities. Although the patient could stand with the eyes shut, she walked with an uncertain step unless her eyes were directed to the ground, as is the case in locomotor ataxia. She could not put the finger on any given part of the face unless she had her vision to guide her, and even with that assistance she did not readily and with certainty direct the movements of the hands.

The other special senses besides the touch, which, as I have said, was markedly lessened in acuteness, were all more or less deranged. Ophthalmoscopic examination showed the existence of neuro-retinitis in both eyes; objects looked blurred, and were generally apparently surrounded with a halo. Occasionally she had had momentary double vision. The pupils were equal in size, but extremely slow to respond to an increased or diminished amount of light.

The hearing was diminished in acuteness. With the left ear she could not hear the ticking of a watch at a greater distance than twenty inches, and with the right ear, twenty-six inches. The tuning-fork placed on the forehead was heard, but the sound was not intensified when the meatus was closed. On the contrary, it seemed to be lessened. I was, therefore, of the opinion that the auditory nerves were affected. The eustachian tubes were pervious.

At one time there had been tinnitus, but latterly this had disappeared. There was no impaction of cerumen, and the drum-heads were apparently healthy.

The senses of taste and smell were markedly diminished in power, the latter being almost entirely abolished. The lining membrane of the mouth and fauces had lost a great deal of its normal sensibility. Thus, she could not, by the taste or the feeling, from the contact with the tongue and mucous membrane, distinguish a clam from an oyster or fish from roast beef.

The mental phenomena were not less strikingly exhibited. There were frequent hallucinations, both of sight and hearing, and delusions that attempts were being made by certain Frenchmen she spoke of to injure her with oil of vitriol, which, she declared, they put into the bed in which she slept and the food she ate.

There was manifest deterioration of the mental power. In answering the simplest question she looked fixedly at the interrogator for fully a minute before speaking, apparently not comprehending its purport, or else uncertain what reply to make. Some quite simple matters she evidently did not understand at all. Thus she could not tell me how much sixty and twenty-five made; and when I asked her what a book was made of, she fixed her eyes on me for some time and finally said, "Oh, all those things," and I could get no other answer out of her.

Her memory was equally weakened. She required much prompting before she could tell where she lived, and made several errors, which, however, she corrected herself, in giving me the names of her children.

Perhaps her memory for words was slightly impaired, but certainly there was no decided aphasia. She could, without much difficulty, give the names of all articles I mentioned to her, and she exhibited no other evidence of defective articulation than that due to paresis of the tongue.

She slept badly, often awoke startled, and was pacified with difficulty.

The hallucinations to which I have referred were not fixed. Those of hearing consisted of human voices telling her how the "Frenchmen" were going to proceed against her, and of the "Frenchmen" themselves abusing and threatening her. Those of sight were of entirely different objects, for, strange to say, she never saw the "Frenchmen." They consisted generally of apparitions of friends who had long been dead, and were most frequent in the afternoon and evening.

When I add that her appetite was bad, that her bowels were constipated, that the urine contained a large excess of urates, without other abnormality, that the pulse was slow and feeble, and that the animal temperature was, in the axilla and under the tongue, never above 96°

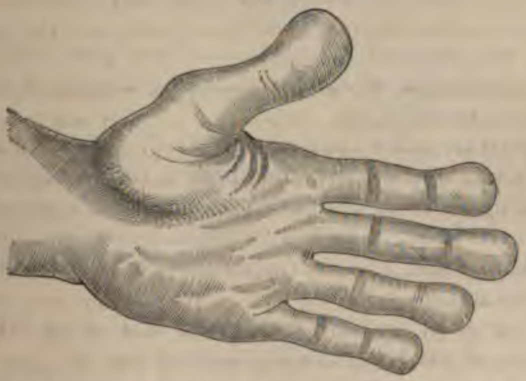
Fahr., and often half a degree below this, I have given as full an account of the symptoms as is necessary for a full understanding of the case.

I saw nothing more of this patient till December 11, 1880, when I again subjected her to careful examination. I then ascertained that the temperature in the axilla and under the tongue had fallen to 94.8°, and that the electric contractility of the muscles to both the galvanic and faradaic currents was markedly lessened in all parts of the body. Generally, the disease had advanced. The strength was reduced, the turgidity of the face and limbs had increased, and the sensibility of the skin was more impaired than when I last saw her. In addition, the vision and hearing had become so much affected that she was almost blind and deaf.

As regards the mental symptoms, there had been no advance, and in some respects a slight degree of improvement had taken place. Thus, while her mind appeared to be fully as sluggish as when I first saw her, the hallucinations and delusions which were then present had disappeared, and no others had taken their place. In fact, she had forgotten all about the "Frenchmen" who were formerly such causes of discomfort to her.

Her fingers (Fig. 26) were more enlarged at the extremities than they were when she was last under my charge, and I discovered that her toes were in a like condition. The tongue, which at former examinations exhibited no evidence of departure from the normal appearance, was now decidedly swollen, and the speech was consequently more labored and indistinct.

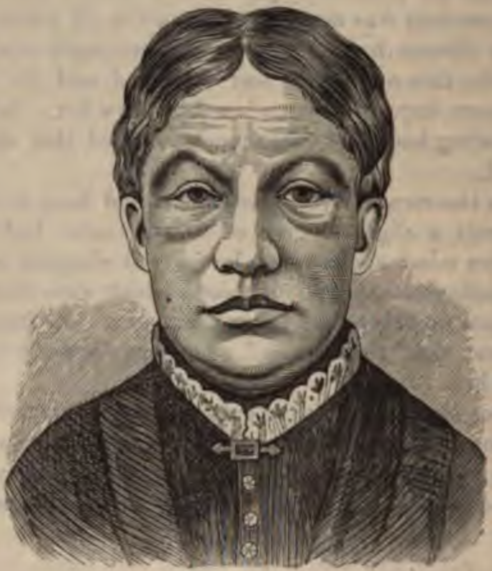
FIG. 26



The urine was of 1018 specific gravity, and was free from albumen and sugar.

But while writing this chapter (January 7th) a second case has come under my observation, differing in no essential respect, except as regards the stage of the disease, from the one the details of which have just been given. Of this instance I am enabled to present a portrait taken from a photograph (Fig. 27). The patient—a female, aged thirty-three—constitutes what I should consider a typical case of myxœdema. With her, as in the other example, the mental symptoms began before any swelling was observed in the face or other part of the body, and consisted of depression of spirits amounting almost to melancholia. There are as yet no delusions.

FIG. 27.



The temperature under the tongue is 95.5°. There is a good deal of irregular action of the heart, and there is very persistent insomnia.

The swelling is more noticeable about the face and neck than in other parts of the body. The fingers are, however, beginning to show the "spade-like" form, and the appearance of œdema is noticeable about the arms and chest.

The further consideration of this case is deferred till I have had the opportunity of studying it with thoroughness.

Causes.—*Sex* appears to be as trong predisposing cause, for, of all the cases observed, only two have been observed in males. One of these, and it is somewhat doubtful if this was a true instance, occurred in the experience of Dr. Savage, the other in that of Dr. Inglis. Age is also a determining predisposing cause, for all the cases have been observed in persons who have reached middle life.

Pregnancy has been thought to exercise a predisposing influence over the causation of myxœdema, but, as I think, without sufficient reason. As to the immediate or exciting causes, nothing is known.

Diagnosis.—Myxœdema is not a disease of difficult recognition. The mental phenomena and the peculiar swelling of the body will of themselves serve to diagnosticate the disease from any other. This œdema, unlike that due to the accumulation of serum in the cellular tissue, does not pit upon pressure, but is, on the contrary, resilient, just as is a rubber ball filled with air. The clinical history will serve to distinguish the swelling of the face from the like condition induced by large and continued doses of arsenic, and the clubbed fingers from the similar formation attendant upon those cardiac affections which interfere with the return of the blood to the right side of the heart.

In scleroderma there is a similar swelling of parts of the body, due to hypertrophy of the skin, but in this affection the surface is hard, and there is a sensation of tightness about the parts involved which is not present in myxœdema. Moreover, there is no permanent reduction of the temperature of the body in scleroderma, as is met with in myxœdema, and there are no mental symptoms, such as form so strong a feature of the latter disease. Scleroderma is a disease of a much younger period of life than is myxœdema, most of the cases observed having been under thirty-five years of age.

Notwithstanding, however, these marked points of difference, it is quite probable that the two affections have been confounded.

Prognosis.—The prognosis is bad. Several cases have terminated fatally, and in no one has there been any amelioration from treatment.

Morbid Anatomy and Pathology.—In regard to the connection of the phenomena with the morbid anatomical condition to which reference has been made, two views have been expressed.

Dr. Ord regards the symptoms as being directly due to the fact that the peripheral terminations of the nerves are so surrounded and compressed by the mucoid tissue deposited about them that they are prevented receiving impressions in their full force, and that, hence, the central organs of the nervous system act less energetically than when excitations reach them in full force.

That this view may be correct, is, I think, exceedingly probable, but the symptoms can not all be accounted for in that way. It certainly will not explain the occurrence of hallucinations and delusions, or the periods of maniacal excitement which have been observed in some cases. It is true, as Dr. Savage admits, that delusions have been ascribed to the annihilation or reduction of peripheral sensations, and it is equally true that the blind may have hallucinations of sight, or the deaf hallucinations of hearing; but in all such cases, as well as in those of general paralytic insanity in which anæsthesia is present, there is ample reason for believing in the existence of central disease.

I must, therefore, agree with Dr. Savage in the opinion that the mental symptoms are the result of primary brain disease, probably to the deposit of the mucoid tissue around the cells of the nervous centres. In the case which I have had the opportunity of studying, there were symptoms of intellectual derangement before any swelling of the body or limbs was observed, and before any sensory disturbances occurred.

In the cases in which post-mortem examinations have been made (those of Dr. Ord) the mucoid deposit was found in abundance throughout the brain as well as in almost every other part of the body so that there is no improbability in the suggestion that the morbid process may begin there.

At the same time, the padding to which the nerves are subjected must certainly interfere with their healthy functionation, and hence I think it quite reasonable to hold the view, as I do, that the phenomena of myxœdema are the result both of central and peripheral disturbances.

Dr. Ord's researches did not extend to the minute examination of the nervous elements of the brain and spinal cord, and hence we are in ignorance what change, if any, is effected in the cell-structure of these organs. It is extremely probable, however, that the starting-point will be found to be in the ganglion cells, just as it is in those diseases of the spinal cord, the morbid anatomy of which has been so thoroughly studied within the last two years.

Treatment.—Nothing appears to have been of any material service in the treatment of myxœdema. Electricity in all its forms has been tried without effect. In the first case which came under my own observation I prescribed phosphorus and strychnia, with the view of stimulating the nerve-centres and improving their nutrition. With the same object, a full and nourishing diet was enjoined. The treatment was only followed for about a month, the patient falling into the hands of an irregular practitioner, who treated her for dropsy. At the last visit to her I prescribed arsenious acid in doses of $\frac{1}{25}$ grain three times a day; and this was the treatment, in conjunction with such hygienic measures as were calculated to build up the organism, which I gave to the second case.

CHAPTER XVII.

CEREBRAL SYPHILIS.

Although the relations of syphilis to diseases of the brain have been considered in the foregoing chapters, it seems advisable to treat the subject with more particularity under its own special head. In so doing I shall avail myself to a great extent of the excellent epitome the matter by Dr. Labadie-Lagrave, in the appendix to the French edition of this work.

ANATOMICAL LESIONS.

a. Neoplasms—Hyperplasia of the Connective Tissue.

At the autopsy of individuals who, in the course of a constitutional syphilis, have presented brain troubles, the lesions which are discovered in the intra-cranial organs for the most part consist of syphilomata or gummy tumors. As Virchow has stated, these may be either hyperplastic forms by excess of normal growths, or they may be hetroplastic—that is, constituted of substance not normal to the position in which it is found.

The neoplasm is sometimes diffused and sometimes more or less exactly circumscribed. According to my experience, the latter is much the more common form. It may present to the naked eye two different aspects, and both of these may exist in the same subject. Thus sometimes the new growth is of a gelatiniform consistence, of a more or less grayish-red color, and is lost imperceptibly in the surrounding tissues. Again, it forms a dense mass of cartilaginous consistence, brittle, and showing, when a section is made through it, a homogeneous caseous appearance. These yellowish-colored masses are entirely isolated and completely circumscribed. They are provided with a fibroid covering.

Sometimes they are found as small striated nuclei scattered through the reddish gelatiniform exudation.

Of these two varieties of syphilomata, the second seems to be derived from the first. It is formed of a basis of connective tissue, more or less altered, in the meshes of which are perceived nuclei and rounded cells. It is probably as a consequence of atrophic degeneration of their elements that the soft and reddish masses are transformed into tumors of a yellowish color and of considerable consistence. Syphilomata of the nervous centres have special predilection for two intracranial situations: the dura mater and the sub-arachnoid space. According to M. Fournier,[1] cerebral gummata are, in the great majority of cases, peripheric—that is, they are located in the cortical layer of the hemispheres. It is rarely the case that they are found in the central regions, and, when this is the case, it is almost always the gray substance—optic thalamus and corpus striatum—that they select. They are also much more commonly found in the anterior region of the brain than in the posterior, and at the base, especially its middle portion, than at the vertex.

When they have their point of departure in the dura mater, they are developed between two thin laminæ of this membrane, and their size varies from that of a pigeon's egg to that of a hen's egg. The action of such a growth upon the brain is that of simple compression.

---

[1] "La syphilis du cerveau," Paris, 1879, p. 57.

When, however, the neoplasm is developed primarily in the subarachnoid space, it invades all the organs (vessels, nervous tissue, etc.) by which it is surrounded, pressing the pia mater before it into the substance of the brain. The greater number of syphilomata met with in the tissue of the brain have this origin. The neoplasm may, moreover, lose its circumscribed form and assume the character of a diffused infiltration. It is very rarely that it appears as miliary nodosities situated in the dura mater or the other membranes. Engelstedt has published a case of this kind, as have also Leon Gros and Lancereaux.

Aside from these hyperplasiæ it does not appear that syphilis has power to produce an encephalitis passing on to softening or suppuration. A syphilitic caries of a cranial bone may give rise to a suppuration which is propagated to the dura mater and cerebral tissue, or a gumma or an alteration of arterial vessels, such as will presently be considered, may be the point of departure for a formation of pus or a simple softening. But, judging from the minute analysis of facts adduced by Heubner,[1] it has never been demonstrated that, taking these circumstances out of the question, there is ever developed a true syphilitic encephalitis.

b. Syphilitic Alterations of the Arteries of the Encephalon.

The alterations which syphilis may produce in the arteries of the encephalon are numerous. It is only recently that they have attracted the attention of pathologists, and are even yet very imperfectly known.

In 1863 Wilks pointed out the existence of a gummy arteritis, of which since then the macroscopic characteristics have been well studied by different English authors, especially by Dr. Hughlings Jackson. This latter observer noticed, as did also others, that gummy arteritis is often the cause of thrombosis and softening of the tissues of the encephalon.

Quite recently a German author, Heubner, has described a new variety of syphilitic arteritis. The lesions which characterize it are seated immediately under the inner coat of the artery, between the endothelium and the fenestrated membrane. They involve, therefore, the more vascular part of the arterial wall. They consist, in the beginning, of an active proliferation of the cellular elements of the endothelium, of which the products constitute nodosities which raise the internal coat of the artery, more or less obliterating the calibre and giving rise to thrombosis. The neoplasm sometimes becomes vascular and forms a veritable neomembrane under the endothelium, and sometimes undergoes inodulary retraction, causing narrowing or even complete closure of the vessel.

[1] "Die luetische Erkrankung der Hirnarterien," Leipzig, 1874.

In addition, MM. Charcot and Pitres have shown, by the autopsy of a woman who at the time of her death exhibited undeniable syphilitic cutaneous manifestations, the alterations produced by nodulated periarteritis in the arteries of the encephalon.

The several steps in the morbid process as it affects the cerebral arteries are far from being known. M. Hanot, in a recent work on the subject, has well put the questions: What are the relations in evolution between gummata and the endarteritis of Heubner? Must we admit that this arteritis is definitely established? Are we warranted in placing periarteritis near it as another form of syphilitic arteritis? These are questions which future researches will not fail to elucidate.

c. *Syphilitic Meningitis.*

Under the heads of "Chronic Verticalar Meningitis" and "Chronic Basilar Meningitis" the relations of syphilis to inflammation of the membranes of the brain have been considered with some degree of fulness, and there is accordingly not much to say in this place.

We have seen, too, that the great majority of gummata within the cranium have their origin in the membranes, and particularly in the dura mater and the subarachnoid tissue. According to M. Alfred Fournier, there can also be developed under the influence of syphilis a pachymeningitis and a hyperplasic piameritis, differing in no essential respects from the ordinary inflammations of the dura and pia mater, so far at least as their histology is concerned. Heubner, however, denies that these conditions ever exist. According to the German author, there is no case on record in which the altered membranes were submitted to rigid microscopical examination with the result of demonstrating the existence of this simple hyperplasic inflammation in cases of cerebral syphilis. To the naked eye, the remains of a gummy meningitis may present some signs of such a disease, but the microscope can alone afford satisfactory evidence of their real nature.

Sometimes there are adherences between the membranes themselves, and again adhesions of the membranes to the cortical substance of the brain. Indeed, whenever the pia mater is the seat of syphilitic inflammation, it can not be separated from the cortex without violence and resultant tearing away of the gray tissue.

Syphilitic patients have died after having exhibited grave cerebral symptoms, and in whom after death no lesions could be found. Heubner mentions several such cases, and two have come under my own observation.

Besides the growths of the brain or its membranes, it must be borne in mind that the endocranium may be the seat of the morbid formation. In his excellent work on cerebral syphilis, Dr. Dowse[1] gives two cases of what were probably instances of this kind; and most physicians whose practice throws them in the way of seeing cases of bra syphilis have witnessed others similar. Subsequently[1] Dr. Dowse say

---

[1] "Syphilis of the Brain and Spinal Cord," London and New York, 1879, p. 18.

"In the examinations which I have made of the brain after dea (over one thousand), I have been surprised to find in how small a nu ber this disease appeared to originate in the under layer of the pe osteum of the endocranium. I think this may perhaps be account for by the fact that where a gumma of the inner table of the skull do arise, the clinical features as evidenced by pain, etc., are so marked (f these manifestations usually occur with the existence of external gu mata) that remedial measures are adopted early, and thus promot absorption before the membranes of the brain become involved."

ETIOLOGY.

The cerebral manifestations of syphilis are very common. They may show themselves at the beginning of the secondary period, but generally a much longer time—often many years—after primary infection elapses before they appear. Certain circumstances favor the development of cerebral syphilis. Among them are bad specific treatment, preëxisting nervous affections, emotional disturbances, excesses of all kinds, and generally every cause capable of weakening the nervous system. Quite recently I had a case under my charge in which the patient, a gentleman fifty years of age, was suddenly attacked with pain in the head, vertigo, and paralysis of the left third nerve, immediately after a period of great excitement in Wall Street. Upon inquiry I ascertained that before his marriage, twenty-five years previously, he had been treated for a hard chancre, but had never had any symptoms of constitutional syphilis except a cutaneous eruption during the first year after infection. Under the use of mercury and iodide of potassium the brain symptoms entirely disappeared in a few weeks.

Virchow has expressed the opinion that the localization of syphilitic manifestations depends in many cases on noxious external influences. Thus we sometimes see the appearances of such essential phenomena soon after the inception of an injury of the cranium; and it has long been thought that hydrargyrum might be the cause of an inflammation or softening of the encephalon.

Age is without special influence in the development of cerebral syphilis, and the same may be said of sex.

GENERAL SYMPTOMATOLOGY.

The variety in the situation and in the nature of syphilitic lesions of the encephalon causes great differences in their symptomatology. A prodromatic sign of much value on account of its constancy is headache. This symptom is always worthy of great attention, for it often precedes for a long time the appearance of more grave manifestations; for it is of great importance to prevent the development of conditions which it may be difficult to remove. "It is everything," says M. Fournier, "to recognize cerebral syphilis in its beginning and to discover its origin."

---

[1] *Op. cit.*, p. 104.

According to this judicious observer, headache, when prodromatic of cerebral syphilis, presents itself under the three following types:

1. Severe pain, with a sensation of weight.
2. Constrictive pain, seeming to the patient as though the head were about to split open.
3. Pains as if from blows with a hammer, instantaneous and extremely severe.

These several forms of headache may be met with in the same patient. Generally they are particularly manifested during the night, as is the case with osteoscopic pains. Without medical treatment, they may disappear after a few months to return again spontaneously. It is rarely the case that the pain occupies the whole head, being either unilateral or limited to the anterior or posterior region. It may even occupy a very circumscribed spot, and then presents all the characteristics of the *clavus hystericus.* According to M. A. Fournier, "a violent and intense pain in the head, with nocturnal exacerbations of long duration, chronic, and frequently recurring, is a symptom that almost invariably indicates the existence of syphilis, and which should always excite suspicion."

It is proper to insist upon the following point: Headache is regarded generally as an essentially prodromatic symptom in the sense that it has for its cause in many cases lesions of the walls of the cranium, and appears first at a time when the intracranial organs have not yet suffered morbid change (Hueter). Later, when the dura mater is involved, the headache becomes more obstinate, more fixed, and is then no longer a prodromatic symptom. It is cerebral syphilis itself.

Among the other symptoms, insomnia must be placed in the front rank. It may depend directly upon the cephalalgia, but is often observed when there is no pain in the head. This symptom especially demands attention when it is met with in young subjects.

Among the other phenomena are vertigo and sensations of faintness, a feeling of weight in the head, failure of memory, difficulty in concentrating the attention and forming ideas, and even aberration of the faculty of speech, either as a forgetfulness of words or an embarrassment in pronouncing them properly. Sometimes there are great intellectual and moral depression, and, again, there is undue mental exaltation. These conditions may be combined in the same individual, alternating with each other; often they are so slight as not to attract the attention of the patient, who does not consult a physician till some more grave symptom, such as an epileptiform seizure, alarms him.

Such attacks may supervene, while there is every appearance of excellent health, and may recur at distant intervals. In general, the later they are in appearing, the more persistent they are in remaining. Sometimes the paroxysm is not to be distinguished from one of true epilepsy; at other times it lacks the initial cry or the convulsive movements, or is limited to one side of the head, as in symptomatic epilepsy. Quite often the convulsive crisis is followed immediately by the phenomena of motor paralysis. A syphilitic epilepsy may only be manifested under the form of the *petit mal.*

M. Alfred Fournier has insisted, in his lectures on this subject, that in many cases of syphilitic epilepsy there are no pathognomonic characteristics distinguishing the attack from one of common epilepsy—that it is an error to assert, as have some authorities, that in syphilitic epilepsy the paroxysms are particularly apt to occur in the night, and that they follow each other with great rapidity for a while, and then cease for a long period. The only symptoms, according to him, are the following:

The convulsive phenomena are complicated with other cerebral manifestations, such as paralysis of a cranial nerve or optic neuritis, which persists during the intervals between the attacks.

The convulsions, instead of beginning in early life, do not appear till after puberty, and then subsequently to a constitutional syphilis, which has arrived at its secondary period.

It is certainly true, as Mauriac[1] declares, that epilepsy is not generally among the earlier manifestations of cerebral syphilis. Nevertheless, I have seen several cases in which an epileptic paroxysm was the very first evidence that the brain was involved. Mental troubles sometimes then appear as depressing feelings, consisting of a kind of intellectual torpor, which advances slowly, in which there is notable weakening of the memory, and which finally terminates in melancholia or mania. At other times the psychical phenomena pursue a rapid course, and consist of periods of great excitement, variability of character and disposition, hallucinations, and furious delirium. These troubles of the intelligence may degenerate into complete dementia. Bell has reported a case of insanity which existed in a syphilitic patient, which lasted two years, and was then cured by a mercurial treatment.

It sometimes happens that cerebral syphilis is manifested under the form of general paralysis of the insane, though sometimes, as Wilks has asserted, the *delire des grandeurs* is absent. Fournier is of the opinion that while general paralysis may be developed in a syphilitic lesion, it has not been demonstrated that syphilis exercises any influence in the production of this disease. According to the eminent physician of the Saint Louis Hospital, of Paris, the affection which some have taken for general paralysis of the insane is only a common general paralysis, which admits of cure by proper anti-syphilitic treatment.

---

[1] "Mémoire sur les affections syphilitiques précoces des centres nerveux," Paris, 1875, p. 162.

However all this may be, it is certainly true that general paralysis of the insane is not infrequently developed in the course of constitutional syphilis, and that there is just as much reason for regarding it as of syphilitic origin as epilepsy, or other manifestations of the disease, as it affects the brain.

Aphasia.—Troubles of speech are very frequent in the course of cerebral syphilis. They present themselves under variable aspects, sometimes being of early appearance and again not coming till late. They may consist of only a slowness of speech, the articulation being explosive or staccato, as is the case in disseminated cerebral sclerosis; at other times there is difficulty in pronouncing words or associating them in a sentence; and again there is true aphasia with agraphia, and this symptom coincides with the occurrence of lesions in the vicinity of the third frontal convolution, such as gummata, meningeal induration, and syphilitic arteritis with softening. It often happens that the aphasia disappears with as much suddenness as it is developed, in which case it is probably due to congestion in the course of the speech tract. Not long since I reported[1] an interesting case of this kind; another is detailed in the present treatise (page 244), several have been given by Tarnowsky,[2] and Fournier[3] declares that when it occurs as an early manifestation of cerebral syphilis it is almost always ephemeral.

Motor Paralysis.—The cortical lesions which give rise to aphasia frequently involve the neighboring motor zone. It is therefore not surprising to find syphilitic aphasia complicated with a more or less complete motor paralysis of the right side. But the paralysis due to syphilis of the brain may of course appear on either side of the body.

It is rarely complete in the beginning. It advances slowly, with periods of amendment and of aggravation, and is complicated with convulsive seizures. It has, in fact, all the characteristics of cortical paralyses. It must be borne in mind that syphilitic lesions of the encephalon are most commonly met with in the membranes and in the cortical substance of the hemispheres (twelve out of fourteen, according to Jaksch). The paralysis of the limbs is often preceded by that of a cranial nerve. Thus, for example, a patient who is apparently in perfect health suddenly becomes the subject of ptosis, strabismus or diplopia, and at other times of a diminution of visual power. Again, the muscles of the face are the seat of temporary contractions, or a

---

[1] "Syphilitic Aphasia; Neurological Contributions," No. 1, vol. i, New York, 1879, p. 62.

[2] "Aphasie syphilitique," Paris, 1870.

[3] *Op. cit.*, p. 242.

CHAPTER XVIII.

SYMPTOMATOLOGY OF CEREBRAL LESIONS.

I.

CORTICAL PARALYSES.

For a long time it was believed that the different regions of the cortical substance of the brain were endowed with the same functions, and that thought, memory, volition, and perception had a common relation to all parts of the gray substance forming the external surface of the convolutions. Flourens, studying the effects of partial disturbance of this gray substance, arrived at the conclusion that the results were the same whatever was the seat of the lesion.

In 1864, as has already been brought to the notice of the reader, the researches of Broca demonstrated the existence of a special centre for language—a fact which Bouilland and Dax had previously done much to establish. This centre was shown to be situated either in the posterior part of the left third frontal convolution, or in the corresponding part of the right third frontal convolution, according as the individual was right- or left-handed. For a long time no further progress was made in the direction of localization, and no one ventured to extend the discovery of Broca and to apply its principles to other functions, the seat of which, all agreed, was in the gray substance of the hemispheres.

In 1870, the German physiologist Hitzig, applying a galvanic current to the gray substance of the hemispheres—until then regarded as inexcitable—saw, to his great astonishment, muscular contractions produced in the opposite side of the body. Moreover, the galvanic excitation of the same part on the surface of the hemispheres always produced contractions in the same group of muscles. Such, in fact, were the relations between the irritated region of the cerebral cortex and the groups of muscles which contracted under the influence of the excitation, that Ferrier, repeating on a monkey the experiments of Hitzig, before the Royal College of Physicians of London, has been able to predict what group of muscles he would cause to contract.

These experimental results of Hitzig and Ferrier have caused a complete revolution in our views relative to the physiology of the brain, for they have shown that the gray substance forming the periphery of the hemispheres is not, as had been thought, inexcitable. And, on the other hand, the question of cerebral localization has received a degree of elucidation which has already led to a vast amplification of our knowledge of cerebral physiology and pathology. It is therefore scarcely a matter of doubt that the several groups of muscles of the body are in direct anatomical and physiological relation with as many ideomotor neuralgia of the fifth pair is developed. These phenomena, which may disappear and reappear many times, generally precede by several months, or even years, paralysis of the limbs.

It is rarely the case that paralysis of syphilitic origin begins with an apoplectiform seizure. When this does occur, the apoplectic attack is generally the immediate result of some strong emotional disturbance or an alcoholic or venereal excess. In such cases the resemblance to hemiplegia from cerebral hæmorrhage or embolism is complete; but when it is complicated with decided syphilitic manifestations its specific origin is extremely probable. M. Fournier asserts that seventy-five out of every one hundred cases of ocular paralysis are syphilitic; but it is probable that the proportion is not nearly so great as this, and, with M. Charcot, we must recognize the fact that many cases of such paralysis are the initial phenomena of locomotor ataxia.

Troubles of vision are very frequent in the initial period of syphilis. According to M. Fournier, both eyes are generally affected, and the morbid process advances very rapidly. All degrees of weakness of vision, from a slight amblyopia to complete blindness, are observed. I have a case now in mind, which I saw in New Jersey a year ago, in which the blindness was so intense that the passage of a bright light before the eyes was not noticed, and yet in less than two months, under the use of large and increasing doses of iodide of potassium and the moderate use of mercury, the sight was entirely restored. Generally these visual troubles are complicated with other phenomena, such as headache, convulsive seizures, etc. They depend upon optic neuritis, which offers to the ophthalmoscope no essential differences from the non-specific form of the affection.

Derangements of the sense of hearing are also common among the early symptoms of cerebral syphilis. They are probably due to inflammation or congestion of the auditory nerves, and usually are met with in both cases. Like the visual troubles from like cause, they are, if not of too long duration, amenable to treatment, and sometimes disappear with great suddenness.

The *duration* of cerebral syphilis depends upon the nature of the encephalic lesions and upon the treatment directed against it. Affections of the arteries are the gravest in character, since they may result in an apoplectic attack due to a thrombosis of one or more of the large vessels. Under such a circumstance the patient dies in a few days in a state of complete coma. Whatever may be the lesion, death may occur, and in fact does often supervene. On the contrary, a cure may be almost certainly obtained, more or less complete in character, when the proper treatment is initiated early and carried out with efficiency.

CHAPTER XVIII.

SYMPTOMATOLOGY OF CEREBRAL LESIONS.

I.

CORTICAL PARALYSES.

FOR a long time it was believed that the different regions of the cortical substance of the brain were endowed with the same functions, and that thought, memory, volition, and perception had a common relation to all parts of the gray substance forming the external surface of the convolutions. Flourens, studying the effects of partial disturbance of this gray substance, arrived at the conclusion that the results were the same whatever was the seat of the lesion.

In 1864, as has already been brought to the notice of the reader, the researches of Broca demonstrated the existence of a special centre for language—a fact which Bouilland and Dax had previously done much to establish. This centre was shown to be situated either in the posterior part of the left third frontal convolution, or in the corresponding part of the right third frontal convolution, according as the individual was right- or left-handed. For a long time no further progress was made in the direction of localization, and no one ventured to extend the discovery of Broca and to apply its principles to other functions, the seat of which, all agreed, was in the gray substance of the hemispheres.

In 1870, the German physiologist Hitzig, applying a galvanic current to the gray substance of the hemispheres—until then regarded as inexcitable—saw, to his great astonishment, muscular contractions produced in the opposite side of the body. Moreover, the galvanic excitation of the same part on the surface of the hemispheres always produced contractions in the same group of muscles. Such, in fact, were the relations between the irritated region of the cerebral cortex and the groups of muscles which contracted under the influence of the excitation, that Ferrier, repeating on a monkey the experiments of Hitzig, before the Royal College of Physicians of London, has been able to predict what group of muscles he would cause to contract.

These experimental results of Hitzig and Ferrier have caused a complete revolution in our views relative to the physiology of the brain, for they have shown that the gray substance forming the periphery of the hemispheres is not, as had been thought, inexcitable. And, on the other hand, the question of cerebral localization has received a degree of elucidation which has already led to a vast amplification of our knowledge of cerebral physiology and pathology. It is therefore scarcely a matter of doubt that the several groups of muscles of the body are in direct anatomical and physiological relation with as many ideomotor centres, occupying each its distinct position in the anterior regions of the gray matter of the convolutions. After performing a great many experiments, Hitzig arrived at the conclusion that the exact situation of each one of these centres was a matter of certainty. He assigned to them the ascending frontal convolution as common property. The superior part of this convolution contains the centres of movements for the lower extremity of the opposite side of the body. Passing downward, we come successively to the centre for the upper extremity, that for the face, and the centre for the movements of the lips and the tongue.

But facts deduced from experiments contradictory of the results obtained by Hitzig were not slow in being brought forward. Sampt, supported by a case in which a cysticucus was situated in the ascending frontal convolution without giving rise to any troubles of motility of the opposite side, denied all connection between the functions of this convolution and voluntary motion. According to Goltz, the effects consecutive to the destruction of a limited portion of the gray substance of the convolutions are not dependent on the seat, but only on the extent of the lesion. These effects would be the same whether the experiment were performed on the anterior or posterior part of the hemispheres, always, however, affecting not only motion, but general sensibility and vision as well. These results of Goltz, however, lose much of their value from the fact that the author, as he himself declares, practiced very considerable mutilations of the animals subjected to experiment. On the contrary, Ferrier, substituting galvanic or faradaic electricity as the exciting agent, found that there was undoubtedly a motor zone in the anterior part of the gray substance of the hemispheres. He, however, limited the extent of this zone to about the anterior two thirds of the surface of the brain; that is to say, to a space ten times larger than the space occupied by the ascending frontal convolution. Ferrier's results have been confirmed by MM. Carville and Duret, who have had recourse to an inverse process, they having studied not only the influence produced by excitation, but also that caused by partial destruction of the cortical substance.

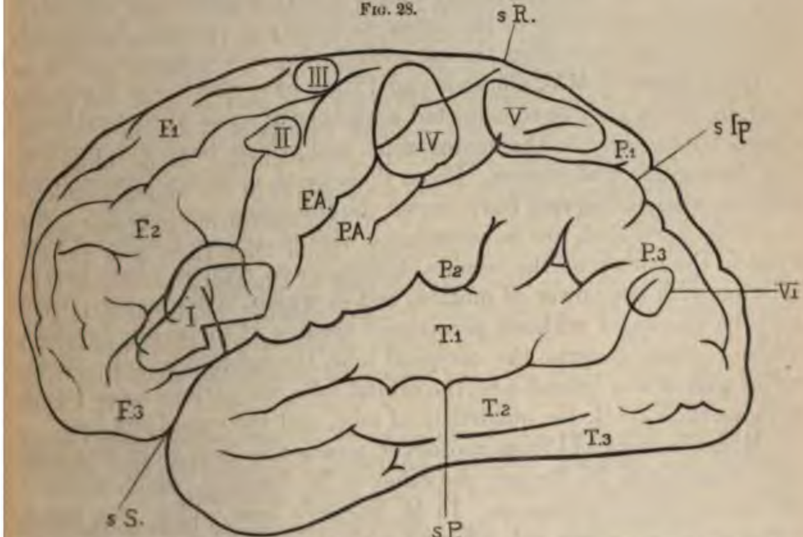
The researches of Betz have given an anatomical basis to the theory of cerebral localizations. This observer, having submitted minute portions of the cortical substance of the hemispheres to microscopical examination, discovered two regions which were distinguished from the rest of the convolutions by the presence of large pyramidal cells measuring $0^{mm}.12$ in their long and $0^{mm}.06$ in their short diameter. The first of these regions embraced the anterior central convolution and the superior part of the posterior central convolution as well as the paracentral lobe. The second occupied a variable extent with different individuals, reaching as far as the point of the occipital lobe. These larger ganglionic cells are particularly abundant about the right paraentral lobe. Betz is of the opinion that of these two regions the ante-ior corresponds to the motor zone, while the posterior is probably the entre for the perception of sensitive impressions. The gray substance f the convolutions appears, therefore, to be arranged and have func-ions corresponding to those of the gray matter of the spinal cord.

If distinct motor centres, each having dependent upon it a certain roup of muscles, really exists in the anterior part of the surface of the emisphere, lesions destructive of the region of the cortex represent-ng one of these centres ought to produce paralysis of the muscles in elation therewith.

This point has lately occupied the attention of pathologists, and it has been ascertained that the evidence supplied by pathological anatomy and clinical observation points still more strongly than the results of experiment to the exactness of Hitzig's conclusions.

Thus MM. Charcot and Pitres have collected in a remarkable work a certain number of cases going to show the relations existing between paralyses and distinctive lesions of the gray substance of the convolutions. In all these cases the seat of the cortical lesion was indicated with the utmost exactness. Hence they arrive at the conclusion that the cortex of the brain contains a motor zone, and that this motor zone

FIG. 28.



The figure indicates the probable situation of the motor centres in man, according to the experiments of Prof. Ferrier performed on the monkey: I, centre for the movements of the tongue and the lower jaws (Broca's convolution). II, centre for the movements of the lips (muscles innervated by the facial). III, centre for the movements of the head and neck (rotation). IV, centre for the movements of the upper extremity. V, centre for the movements of the lower extremity. VI, centre for the movements of the eyes. F1, F2, F3, first, second, and third frontal convolutions. FA, ascending frontal convolution. PA, ascending parietal convolution. P1, upper parietal lobule. P2, lower parietal lobule. P3, angular gyrus. T1, T2, T3, first, second, and third temporal convolutions. SR, sulcus of Rolando. SIP, interparietal sulcus. SP, parallel fissure. SS, fissure of Sylvius.

occupies the paracentral lobe, the two ascending convolutions, and probably also the inferior portion of the three frontal convolutions. In the cases cited by MM. Charcot and Pitres, all cortical lesions, whatever their extent outside of this motor zone, were powerless to cause troubles of motility. On the other hand, destructive lesions, even when of very limited extent, situated within this zone, constantly produced motor disturbance. The paralysis was of sudden origin when the lesion had been suddenly produced, and it was limited to a part only of one side of the body when the lesion was restricted to a part only of the motor zone. MM. Charcot and Pitres, moreover, think they are warranted in concluding, from their study of paralyses and convulsions of cortical origin, that the motor centres for the upper and lower extremities are seated in the paracentral lobe of the opposite side and in the two upper thirds of the ascending convolutions, and that the centres for the movements of the lower part of the face are placed in the inferior third of the ascending convolutions in the vicinity of the fissure of Sylvius, and finally that it is very probable that the centre for isolated movements of the upper extremity is situated in the middle third of the ascending frontal convolution of the opposite side.

Quite recently MM. Charcot and Pitres have published a new memoir, in which they have collected a number of cases of cortical lesions situated exteriorly to the motor zone, and which have not produced any derangement of motion. From these facts, collected from different authors, these observers have drawn the following conclusions, which, it will be perceived, are in accordance with their own results: "There exist in the cortex of the cerebral hemispheres, regions which have no relation with the power of motion, and in which, consequently, lesions may be produced without permanent trouble of the motor functions. These regions comprise the occipital lobe, the sphenoidal lobe, the anterior part of the frontal lobe, the orbital lobe, the parietal lobes (except perhaps their feet), the quadrilateral lobe, and the cuneiform lobe."

MM. Charcot and Pitres, moreover, mention in their work two cases, one occurring to M. Berger, the other to M. Jaccoud, which appear to invalidate their conclusions. In M. Berger's case, which was that of a young girl six years old, affected with right hemiplegia and accompanying convulsions, and in which the autopsy revealed the existence of a *sclerosis limited to the occipital convolutions*, MM. Charcot and Pitres say in regard to this case, "that the anatomico-pathological description is exceedingly meagre, and that it is absolutely bare of details relative to the state of those parts of the brain other than the occipital convolutions, and leaves the field entirely open to speculation."

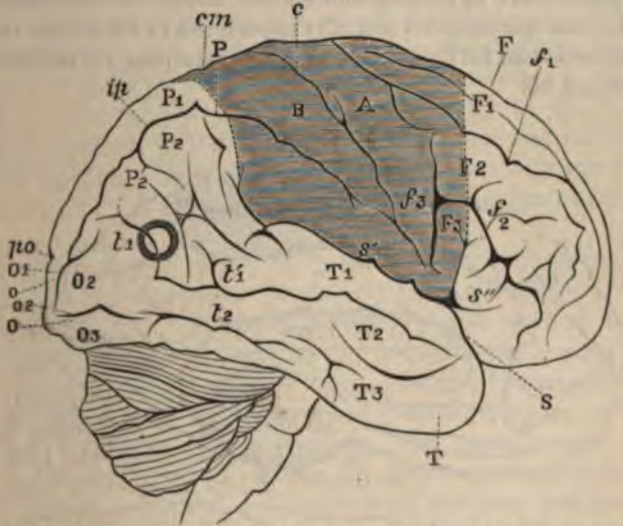
The case of M. Jaccoud[1] is that of a woman sixty years of age, who had been subject to convulsions strictly limited to the right side of the body, a right facial paralysis, flaccid paralysis of the limbs of the left ide, with deviation of the head and of the eyes toward the same side. t the autopsy there were found cortical lesions, involving "the supe- ior parietal lobe, with the exception of the ascending parietal con- olution and the two upper thirds of the angular convolution of the aracentral lobule, the outer wall and the convolution which lies be- ween it and the calloso-marginal fissure, and the convolution of the orpus callosum in that portion comprised between the parieto-occip- tal fissure and the calloso-marginal fissure."

---

[1] *Gazette Hebdomadaire*, 1878, p. 475.

This case presents, according to MM. Charcot and Pitres, some ob- cure points, so far as the anatomico-pathological description is con- erned, and hence it is difficult to determine the precise seat of the ce- rebral lesion. "But it is," they continue, "open to the reproach of being an irregular, singular, and exceptional case, in which a great many anomalies of cerebral symptomatology appear to have been given a local habitation."

Fig. 29.



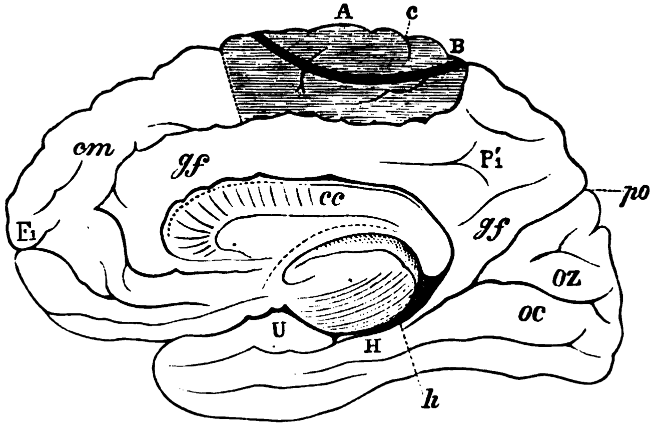
The lesion is shown by the shaded portion. It was a gliomatus tumor, and gave rise to left hemiplegic epileptic seizures and paretic troubles on the left side. On one occasion the head was rotated to the left (case lxvii., thesis of Landouzy). The lesion shown by the circle at the summit of the parallel fissure (meningitis case xii. of M. Grasset, thesis of M. Landouzy) caused ptosis of the left upper eyelid. No other paralytic trouble was noticed.

F, frontal lobe; P, parietal lobe; O, occipital lobe; P_1, P_2, P_3, first, second, and third temporal convolutions; f_1, f_2, first and second parietal fissures; C, sulcus or fissure of Rolando; A, anterior frontal or central convolution; B, posterior central or ascending parietal convolution; F_1, F_2, F_3, superior, middle, and inferior frontal convolutions; f_1, f_2, superior and inferior frontal fissures; f_3, parietal fissure; *ip*, interparietal fissure; *cm*, end of the calloso-marginal fissure; *po*, occipito-parietal fissure; *o*, transverse occipital fissure; o_2, longitudinal occipital fissure; P_1, parietal lobe; P_2, supra-marginal convolution; P_3, angular convolution; O_1, O_2, O_3, first, second, and third occipital convolutions.

Such are the general deductions which MM. Charcot and Pitres have thought themselves warranted in drawing from the whole series of cases brought forward in their memoir. But when one enters into the detailed consideration of these cases, it appears that there are not wanting facts which are opposed to the exactness of their conclusion. The paralysis, for instance, of the same group of muscles may be th result of lesions occupying very different points in the motor zone. To this view the clinical researches of M. Bourdon lend support. In reporting a case of brachial monoplegia of cortical origin occurring in his own experience, this author cites fourteen other cases in which there was a thorough post-mortem examination. From the comparison of the precise seat occupied by the lesion in these different cases, M. Bourdon has arrived at the conclusion that brachial monoplegia may result from lesions of various parts of the motor zone of the hemispheres ; and he has also shown that what is true for brachial monoplegia is equally so for hemiplegia.

M. Louis Landouzy[1] has collected a large number of cases of paralysis consecutive to meningo-encephalic lesions of the fronto-parietal lobe. Of the one hundred and fifty cases cited in his monograph, there were not less than fifty-nine in which the hemiplegia was partial. See Figs. 29 and 30.

FIG. 30.



The lesion indicated by the shaded part of the paracentral lobule represents the extent of a cerebra glioma (case lxvi. of M. Landouzy's memoir). That part of the shaded portion above the curved line represents the internal prolongation of a focus of tuberculous encephalitis (case xxiv. of the same work), which gave rise to paresis and contractions of the left limbs.
A, anterior convolution ; B, posterior central convolution ; *gf*, gyrus fornicatus ; P, præcuneus ; *oz*, cuneus ; H, gyrus hippocampi ; *p*, sulcus hippocampi ; U, gyrus uncinnatus ; *po*, parieto occipital fissure ; *oc*, calcarine fissure.

Nevertheless, it was impossible to draw from this ample material the least definite information relative to the precise localization of the centres in the cortical motor zone for the different groups of muscles affected.

M. Raynaud has observed a case of left brachial monoplegia in an individual in which at the autopsy a single lesion, consisting of a very small *foyer* of red softening, developed on the right hemisphere, and surrounding a meningeal tubercle. This *foyer*, which had not attained to the dimensions of a twenty-five-cent piece, was situated on the ascending parietal convolution at a distance of three centimetres from the superior and internal border of the hemisphere. This case is therefore in opposition to the view of MM. Charcot and Pitres, which locates the probable centre for isolated movements of the upper extremity in the middle third of the anterior frontal convolution.

---

[1] "Contribution à l'étude des convulsions et paralysies liées aux meningo-encéphalites fronto-parietales," Paris, 1876. Thèse inaugurale.

It was also a lesion of the ascending parietal convolution which M. Langlet[1] found at the autopsy of a man forty years old, who, having received thirty-three years before a blow with a boat-hook on the middle parietal region, became subject for the rest of his life to a right hemiplegia with late contractions and atrophy. M. Langlet determined with great precision the seat of the cortical lesion. "The instrument," he says, "had reached the *superior third of the ascending parietal region* at its posterior curve, and had penetrated to the fasciculi of fibres which go to the peduncle externally to the caudated nucleus of the corpus striatum." This cortical lesion had led to a secondary degeneration of the pyramidal fibres, which the author thought he could trace as far as the opposite antero-lateral column of the spinal cord.

In the case of a man observed by M. Prévost, a traumatic lesion of the cranium, with depression of the left parietal eminence, was followed by aphasia with right facial hemiplegia and brachial monoplegia of the same side. The crown of the trephine was applied behind and above the depressed portion of bone. Several fragments of bone were removed, and three of these were of considerable size, and at the same time a few drops of pus escaped. The operation was hardly over before the paralytic troubles and the aphasia had manifestly lessened in intensity. A short time afterward the cure was complete. Experiments made on several cadavers have enabled MM. Prévost and Terrillon to determine with mathematical accuracy the point in the cerebral convolutions which had been injured. They conclude that the depressed piece of bone almost certainly wounded the left ascending parietal convolution, and that it affected, secondarily, the ascending frontal and the second and third frontal convolutions.

M. Lucas-Championnière, who had previously had a case in which he had successfully trephined for a traumatic lesion of the cranium, also has endeavored to ascertain with exactness the region of the cranium which corresponds to the motor zone, and the external points which should guide the surgeon in the operation of trephining. Having determined the course of a line which, drawn on the surface of the cranium, corresponds to the fissure of Rolando, M. Lucas-Champonnière advises that the trephine should be applied to the middle of this line if the hemiplegia be complete, toward the top end if the paralysis be limited to an upper extremity, and toward the lower end if the paralysis affects a lower extremity only.

---

[1] *Gazette Médicale du Nord-Est*, 1877.

The results of the anatomico-pathological researches to which attention has been called, permit us to see that, by following to the letter the precepts formulated by M. Lucas-Championnière, we would be liable to commit many mistakes, and all surgeons do not accept them in their unmodified form. Thus, M. Tillaux has observed a patient who, as a consequence of a blow from a window-shutter received on his head, was affected with aphasia and monoplegia of the right arm. At the post-mortem examination a patch of meningo-encephalitis was found which touched a part of the second convolution and the convolution of Broca. M. Tillaux insists that, in a case like this, the trephine would have led to no beneficial result; and he also declares, and of this there can be no doubt, that a cerebral lesion may produce such remote troubles that the surgeon, by following M. Lucas-Championnière's rules, would be led into error.

In the report made to the Academy of Medicine, by M. Gosselin, on the communication of MM. Prévost and Lucas-Championnière, it is asserted that the surgeon can not judge of the nature of a traumatic lesion of the brain by the functional troubles to which it gives rise. Can we tell whether it is superficial or profound? if it is a laceration or only a compression? Besides, the opening of the cranium constitutes a new danger for the patient, inasmuch as it exposes him to the dangers of septicæmia and meningo-encephalitis. The trephine is especially indicated in cases of fracture of the skull with depression of bone. Then the operation adds nothing to the dangers to which the patient is exposed, while it may at once allow the compression of the brain to be relieved, whether this be due to a fragment of bone or to a hæmorrhagic or purulent accumulation.

It is, therefore, perceived, that surgical observation furnishes no new support to the exact localization of the several motor-centres into which certain physicians are apparently eager to decompose the mass of convolutions.

M. Gosselin,[1] with all the authority which attaches to him, has rightly tempered the enthusiasm of those surgeons who have been too prompt to practice the operation of trephining in accordance with the contradictory results of experimental physiology. On the whole, clinical observation seems to demonstrate that the paralysis of a given group of muscles may depend on lesions of very different points of the motor zone; and also that lesions of a given point of the motor zone may give rise to paralyses of variable situation.

---

[1] "Clinique chirurgicale de la Charité," third edition. Paris, 1878, t. i., pp. 257, 301-333.

Experimental physiology and clinical observation agree that there exists on the periphery of the hemisphere a motor zone, embracing the posterior part of the frontal lobe and the anterior part of the parietal lobe. At the same time, it may be considered an established fact that electrical or pathological excitation of this zone engenders contractions of the muscles of the opposite side of the body, while destructive lesions, involving this zone, produce motor paralysis of the same muscles. But this is as far as we can legitimately go, for in the present state of our knowledge it is impossible for us to deduce from the seat of the paralysis the exact situation of the cortical lesion. Moreover, it is not probable that the motor zone, the existence of which is not a matter of doubt, is divided into a certain number of regions, each one having its distinct function. In other words, it is not yet to be conceded that a given group of muscles is under the exclusive control of a single ideomotor centre located in a determinate part of the motor zone. The different motor cells of this zone are in intimate relation with each other, and they communicate, on the other hand, with motor fibres which go to the contractile organs of the periphery. It is apparent, therefore, that when a portion of the motor zone has been destroyed by a pathological alteration, the muscles which are paralyzed are those which are in anatomical connection with the cells situated in the disordered part of the cortex. Now we have no definite knowledge relative to the course of the motor-fibres in the *centrum ovale*, or of their distribution to the periphery of the hemispheres. There is no demonstration that the fibres destined to a particular group of muscles come from neighboring cells of the motor zone. It is, on the contrary, very probable that there is no such derivation. In fact, one of the characteristics of restricted cortical paralysis, the result of limited lesions of the motor zone of the convolutions, is their transient duration. To reconcile this fact with the theory of localizations, we are forced to admit that the motor centres of the different groups of muscles can be supplied in their functions by other centres. This substitution can not be attributed to the homologous nervous centres of the hemisphere opposite to that which has been injured, for in experiments on the lower animals it has been shown that, after recovery has taken place from a paralysis produced by a cortical lesion, the loss of power can not be reproduced by injury of the homologous motor centres, and further, paralysis disappears when two homologous regions of the motor zones of the two hemispheres have been injured. We must, therefore, concede the point that the several centres of the motor zone of a hemisphere may have their functions substituted, one by the other.

But the matter admits of easy explanation, if we recognize the correctness of the view stated, that the several motor-fibres coming from a group of muscles are distributed to different parts of the motor zone of the opposite side. In fact, the initial paralysis resulting from a limited lesion of the motor zone is due to the circumstance that th lesion destroys the cells whence emanate the *greater part* of the fibr which go to the muscles which are paralyzed—fibres by which the e is accustomed to transmit motor impressions to the group of muscl in question. And we well know how important an influence th of habit is in the education of the different organs of the economy At the end of a certain time, the motor impulsions, which, as a whole, take their origin in the entire extent of the motor zone, follow, in order to reach the paralyzed muscles, the fibres which, while connecting these muscles to that portion of the motor zone which is uninjured, have hitherto remained in a state of functional inertia. It is clear, moreover, that, if the lesion involves a great extent of the motor zone, substitution would be no longer profitable, and the paralysis of cortical origin would persist indefinitely.

What is the function of the cells of the motor zone in the production of those movements which are the result of the excitation of this zone by the electric current? Are these movements the immediate effect of the excitation of the cells? In other words, has the electric current the property of setting in action the cells of the gray substance whence originate the impressions to voluntary movements? Until a recent period, it was generally admitted that the gray substance of the nerve-centres was insensible to our ordinary means of excitation. The experiments of Hitzig seem to demonstrate that the gray substance of the motor zone of the convolutions comports itself differently. But high authorities, such as MM. Vulpian and Schiff, declare that this is in reality an erroneous conclusion; for that it is not by exciting the gray substance which constitutes the motor zone, but rather the subjacent white substance which leads to the production of contractions in the muscles of the opposite side—this excitation, as in the experiments of Hitzig and Ferrier, and others, being caused by the diffusion of the electric current through the gray substance to the white matter below. The facts which demonstrate this diffusion, and the circumstances which favor it, have been well studied by MM. Carville and Duret, and these able investigators have, by the aid of the galvanometer, given direct proof of the diffusion of the electric current through the central substance. Besides this, they have shown that when a weak current, but one of increasing intensity, is applied to a point of the motor zone, nothing is observed but isolated contractions of a group of muscles. These contractions, however, involve little by little all the muscles of the side of the body opposite to the excited hemisphere, and eventually general convulsions ensue. Muscular contractions can still be obtained on the opposite side by exciting the surface of the convolutions outside of the motor zone. The current necessary to produce this effect ought to be proportionally stronger, as the region is farther from this zone.

When, however, the brain is saturated with liquids which are good conductors of electricity, the diffusion of the electricity is greatly facilitated.

When the white tissue subjacent to the motor zone is exposed, it can be directly excited by the electric current, and contractions produced in the muscles of the opposite side of the body. Eckhart has succeeded in following, as far as the corpus striatum, the fasciculi, the excitation of which produced contractions of the muscles of the anterior limb. He did this by performing successive transverse sections of the hemisphere. M. Onimus has seen muscular contractions follow in the muscles of the opposite side of an animal, when, after the removal of a hemisphere, he excited with the electric current the sanguineous liquid, which filled the corresponding side of the base of the skull. The excitation in this case was evidently transmitted by the liquid to the subjacent white substance.

There are still other facts which go to show the diffusion of the electric current in the experiments of Hitzig and Ferrier. Thus it is impossible to understand why the gray substance of the motor zone, if it is really sensitive to electrical excitation, does not respond also to other stimulating agents of which, unlike the electric current, the action is not diffused to the white substance. Moreover, we repeatedly see tumors develop on the surface of the motor zone, by which it must necessarily be irritated, and yet which never give rise to muscular contractions on the opposite side of the body, so long as the gray substance remains intact and the white substance is not exposed. Finally, a number of cases have been published in which there were distinct lesions of the motor zone of the convolutions without there having been paralysis of the muscles of the opposite side of the body.

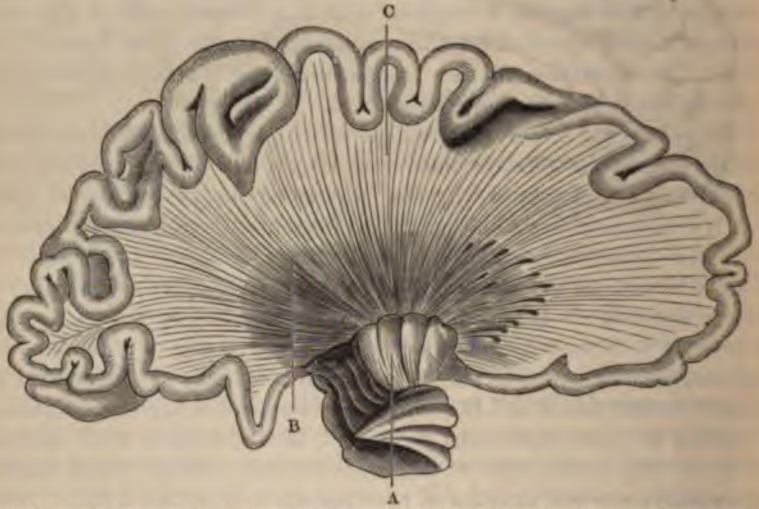
From this standpoint, therefore, it is necessary to distinguish between lesions which only involve the superficial layers of the convolutions and those which at the same time implicate the gray substance and the subjacent white substance. For only in this latter condition is there paralysis of the muscles of the opposite side of the body. Moreover, M. Pitres, in his inaugural thesis, has shown that the lesions which primarily involve the white substance of the centrum ovale, comprised between the internal capsule and the motor zone of the convolutions, are constantly followed by paralysis of the muscles of the opposite side of the body. Now this part of the centrum ovale is only the expansion of the motor portion of the internal capsule. It is constituted, according to the most recent researches, of fibres which directly connect the motor zone of the convolutions with the gray substance of the anterior horns of the spinal cord.

II.

PARALYSES CONSECUTIVE TO CENTRAL LESIONS OF THE HEMISPHERES.

The cerebral hemispheres, attached on each side to a corresponding peduncle, are composed of certain nuclei of gray matter (central ganglia), among which pass laminæ of white tissue, which constitute the greater part of the substance of which each lateral half of the brain is constructed. These gray nuclei are three on each side—the lenticular nucleus, the candated nucleus, and the optic thalamus. The first two are sometimes designated the extra-ventricular and intra-ventricular nuclei respectively, and together constitute the corpus striatum. The candated nucleus and the optic thalamus are separated from the lenticular muscles by a lamina of white substance called the internal capsule, which by its expansion constitutes the corona radiata. These relations are clearly shown in the accompanying diagram of a vertical section of the brain. Now if the fibres of the internal capsule be followed from above downward, it is seen that they come exclusively from the inferior portion of the peduncle. These fibres in reality form two very distinct fasciculi. The one, composed exclusively of sensory fibres, at the posterior and inferior part of the corona radiata, winds round the posterior extremity of the lenticular ganglion, and then turns back to terminate, according to Meynert, in the occipital lobe. This sensory fasciculus, after decussating at the posterior part of the pyra-

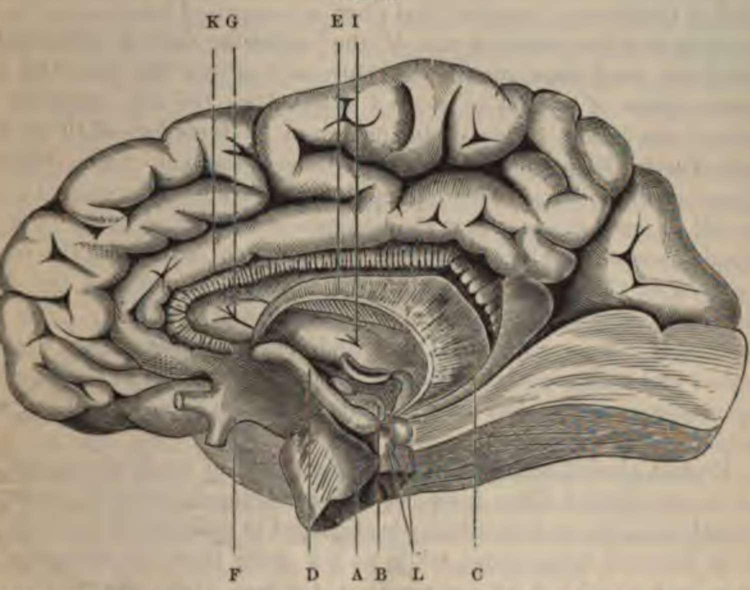
FIG. 31.



Internal capsule, external face. A, radiating fibres; B, foot of the internal capsule; C, internal capsule (external face).

mids, reaches, according to the same author, the posterior column of the spinal cord of the opposite side.

FIG. 32.



Internal capsule, internal face. A, section of the protuberance; B, cerebral peduncle, fibres of the peduncle (passing directly into the optic thalamus); C, fibres from the optic thalamus going to the extra-ventricular nucleus (lenticular ganglion) of the corpus striatum and to the internal capsule; D, postero-external fasciculus, which, starting from the peduncle, passes around the posterior part of the optic thalamus and joins it superiorly; E, fibres going from the optic thalamus to the extra-ventricular nucleus of the corpus striatum; F, chiasma of the optic nerves; G, intra-ventricular nucleus of the corpus striatum; I, optic thalamus; K, convolutions of the corpus callosum; L, tubercula quadrigemina.

The other fasciculus, which constitutes the anterior two-thirds of the internal capsule, consists entirely of motor fibres. These terminate in part in the central ganglia. But the most recent researches demonstrate that the motor portion of the internal capsule contains fibres which extend directly from the inferior part of the peduncle in the corona radiata without any connection with the gray nuclei. These fibres, which are what Flechsig calls *the pyramidal fasciculi, properly so called*, terminate, in all probability, in the motor zone of the cerebral convolutions. Inferiorly they constitute the anterior pyramids, and are continued partly in the posterior column of the lateral opposite half of the spinal cord and partly in the anterior column of the same side.

These statements of the anatomy of the parts concerned are necessary for the full comprehension of the morbid conditions induced by lesions of the different regions of the central mass of the hemispheres.

a. Lesions of the Posterior Third of the Internal Capsule.

Cerebral Hæmianæsthesia.—When a lesion involves the posterior third of the internal capsule, that portion comprised between the optic thalamus and the posterior part of the lenticular nucleus, it is manifested by total hæmianæsthesia, both as regards the skin and the other organs of special sense, and situated on the side opposite to that of the lesion. This is demonstrated by facts furnished by Andral, Türck, Rosenthal, Jackson, Charcot, Vulpian, Luys, Rendu, Veyssière, and others. This general hæmianæsthesia is explained when we call to mind the fact that the lenticulo optic portion of the internal capsule consists of all the sensorial fibres which come from the opposite side of the body. Experimental physiology confirms the data which are supplied by clinical experience and anatomy. The researches of Vulpian, and of Duret and Veyssière, his pupils, demonstrated that experimental lesions of the posterior third of the internal capsule produce complete hæmianæsthesia of the opposite side of the body.

From a clinical stand-point hæmianæsthesia of cerebral origin does not in any respect differ from the like condition induced by hysteria. In both cases general sensibility is abolished in one-half of the body, in all its different expressions, while at the same time the other special senses are abolished or deranged on the same side. As regards the sight, for instance, there is not only a diminution of the power of vision, but there is a concentric and general narrowing of the visual field without there being any change in the normal appearance of the fundus of the eye to ophthalmoscopical examination.

Prof. Charcot has insisted on this fact, as a deduction from clinical observation, namely, that "lesions of the cerebral hemispheres, causing hæmianæsthesia, produce at the same time crossed amblyopia and not lateral hemiopia." German authors, however, in general adhere to the theory of A. von Graefe, that the usual trouble observed in cases of unilateral lesions of the hemispheres is a simple abolition of sight in the corresponding half of the field of vision of both eyes—in other words, a homologous lateral hemiopia. This purely hypothetical opinion is based upon the theory, also hypothetical, of the semi-decussation of the fibres of the optic nerves. Since the time of Newton (1723), it has been admitted that the fibres which come from the optic tracts to form the optic nerves partially decussate in the chiasma. It is also admitted, in order to explain certain pathological facts, that the optic fibres which decussate—that is, those which come from the tract of the opposite side—are situated interiorly after crossing, while those fibres which do not decussate occupy the exterior face of the nerve of the opposite side. It results from this anatomical disposition (which, however, is by no means a demonstrated fact), that the fibres contained in the optic nerve of the left side go to the left half of each retina, and that those which constitute the right optic nerve pass to the right half of each retina (see Fig. 33, page 370).

In order to explain how lesions of the hemispheres produce crossed hæmianæsthesia, giving origin at the same time to crossed amblyopia and not to lateral hemiopia, M. Charcot has proposed a modification of the theory of the semi-decussation of the optic nerves. It is known that the optic tracts, the continuation of which after the decussation constitutes the optic nerves, are in anatomical relation with different ganglia of gray tissue situated at the base of the brain before pursuing their course toward the gray substance of the cortex. The external root of each optic tract, the larger and the more important of the two, is in direct relation with the corpus geniculatum externum, with the inferior portion of the optic thalamus, and with the tubercula quadrigemina anteriora of the same side. Now the distinguished physician of the Salpêtrière is disposed to think that in that part of their traject comprised between the cortical centres and the corpus geniculatum the fibres of the optic nerves complete their decussation. According to this view, the fibres which constitute the optic nerve of one side cross over, some above and others below the chiasma, in such a manner that a lesion exactly limited to one hemisphere concerns only those fibres which reach the retina of the eye of the opposite side.

b. Lesions of the Anterior Two-Thirds of the Internal Capsule.

Cerebral Hemiplegia, Secondary Degeneration, Late Contraction.—Every destructive lesion limited to the anterior two-thirds of the internal capsule causes symptoms of common cerebral hemiplegia, otherwise known as motor paralysis, and affecting the side opposite to the lesion. When the lesion is situated in the anterior two-thirds and posterior third of the internal capsule, we have at the same time the symptoms of motor hemiplegia, associated with the phenomena of hæmianæsthesia described in the immediately preceding section.

A motor hemiplegia consecutive to a *destructive lesion* of the internal capsule is in general very decided, and more or less persistent in character. Moreover, at an advanced stage it is almost invariably complicated with permanent contractions of the paralyzed limbs, as happens in profound lesions of the motor zone of the convolutions and of the subjacent white substance. These contractions are directly due, as already stated, to secondary degeneration of the pyramidal motor fibres, which, crossing from the fronto-parietal region of the centrum ovale of each hemisphere, reach without interruption the posterior part of the lateral column of the spinal cord of the opposite side and the anterior column of the same side by passing through the two anterior thirds of the internal capsule. Flechsig holds that the place of passage of the pyramidal fibres in the internal capsule is the *third posterior quarter*, and that, contrary to the opinion of M. Charcot, it is particularly this portion of the internal capsule which is the seat of the lesion in cases of cerebral hæmorrhage.

The appearance of late contractions is always an unfavorable circumstance in determining the prognosis. It indicates, in fact, that the hemiplegia is due to a lesion of the internal capsule, and that, moreover, our therapeutical measures are powerless against the hemiplegia which has resulted. [At the same time it is not to be doubted that even in these cases ameliorations can be sometimes produced by appropriate treatment.—W. A. H.]

c. Lesions of the Central Ganglia of the Hemispheres.

Transitory Hemiplegia.—Lesions confined to the central ganglia of the hemispheres (lenticular nucleus, nucleus candatus, and optic thalamus) also produce motor paralysis. But this is, in general, of a transient character, and is probably never followed by late contractions.

To explain the non-permanent duration of a hemiplegia confined to the gray nuclei of the hemispheres, it has been said that the different parts of these centres can replace each other in function. Would it not, however, be more natural to admit that the central ganglia do not take a direct part in the execution of voluntary movements? On the one side it is demonstrated that the motor zone of the convolutions originates voluntary motor impulses, and is directly in anatomical relation with the motor centres of the spinal cord. On the other side, there is no case on record in which a lesion limited to the substance of one of these gray nuclei has given rise to secondary degeneration. On the contrary, Türck has noticed a lesion the size of a filbert seated in the body of the nucleus candatus which had not produced a secondary degeneration. In two other cases reported by Türck, a lesion of old date occupied the superior part of the optic thalamus, and in neither of them was there secondary degeneration. This absence of secondary degeneration has likewise been noted by Flechsig in two cases, in which a circumscribed lesion was limited to the external zone of the lenticular nucleus. The central ganglia of the hemispheres seem, then, to be deprived of all direct connection with the direct pyramidal fibres interposed between the cortical motor zone and the motor cells of the spinal cord.

Moreover, to explain the development of transient motor hemiplegia consecutive to lesions in the substance of the central ganglia, we are led to admit that these lesions act only by compression in abolishing for a short time the functions of the pyramidal fibres of the internal capsule whose office it is to transmit voluntary motor impulses.

It is to be noted that the point of selection for cerebral hæmorrhages is the internal surface of the lenticular nucleus. The blood is then extravasated in the space comprised between the external surface and the lamina of white substance which borders it without adhering to it, and which is known as the external capsule. This explains why, in the majority of cases of cerebral hæmorrhage, the hemiplegia is transitory, and is not accompanied by contractions.

Hemichorea of Cerebral Origin.—A few years since, Dr. Weir Mitchell pointed out for the first time that choreiform troubles sometimes complicate the muscular paralysis in patients who have for some time been the subjects of hemiplegia. He gave to this condition the name of post-hemiplegic chorea. More recently M. Charcot has shown the frequent co-existence of hemichorea with hæmianæsthesia of cerebral origin. Explaining this co-existence by the results of a certain number of autopsies, he has demonstrated that the lesions which cause these two varieties of phenomena—hæmianæsthesia and hemichorea—occupy contiguous points of the posterior part of the foot of the corona radiata—the lesions which have been found at the autopsy of patients affected with hemichorea being comprised in the zone which includes the posterior part of the internal capsule, the posterior part of the optic thalamus and of the caudated nucleus, and the anterior tubercula quadrigemina. But M. Charcot is of the opinion that the lesion of the white substance—that is to say, of the internal capsule—is that to which the hemichorea supervening in hemiplegia is to be ascribed. It is to be remembered that this hemichorea is not always post-hemiplegic, but that it may precede an attack of cerebral hæmorrhage. [And it is also to be borne in mind that post-hemiplegic or ante-hemiplegic chorea is altogether a different condition from athetosis, with which, in fact, it has scarcely a feature in common, either as regards the origin, the character of the movements, the accompanying symptoms, or the seat of the lesion. In a preceding chapter athetosis has been fully considered.—W. A. H.]

d. Lesions of the Lateral Ventricles; Ventricular Hæmorrhage.

Convulsions of Central Origin—Early Contraction.—When a hæmorrhagic extravasation breaks through into the cavity of the ventricles, it occasions at the same time coma, paralysis, early contractions, and epileptiform convulsions.

III.

LESIONS OF THE TUBERCULA QUADRIGEMINA—OCULO-PUPILLARY TROUBLES.

It is generally admitted that the tubercula quadrigemina are the centres of origin of the optic nerves. The influence of these organs over the movements of the pupil has been thoroughly demonstrated.

Flourens, for instance, obtained movements of the iris on both sides by exciting the tubercula quadrigemina; and, more recently, Ferrier, by electrizing these nuclei of gray substance, has seen the pupils dilate. According to Flourens, the lesion or ablation of the tubercula quadrigemina of one side produces blindness of the opposite side.

In addition, the tubercula quadrigemina preside over the conjugate movements of the eyeballs. This is shown clearly by the researches of Adamük, published in 1870. This physiologist has shown that the superficial excitation of the anterior pair of the tubercula quadrigemina at different points produces varied movements, but that always both eyes move simultaneously. When the right tuberculum is excited, both eyes deviate to the left; when, on the other hand, the left is excited, both eyes are turned to the right.

These physiological data have found their application in the pathology of the nervous system. In a certain number of cases of the destruction of the tubercula quadrigemina of both sides, complete blindness with dilatation of the pupils has been produced. Thus, in the case of a patient observed in the service of M. Pidoux by M. Blanquinque,[1] and in which during life complete blindness and dilatation of the pupils were present, at the autopsy was found a tumor of the pineal gland, which compressed both pairs of tubercula quadrigemina, especially the posterior pair. In the same patient the eyes were turned downward and to the right. This phenomenon of the conjugate deviation of the eyes is also observed as a consequence of lesions affecting very different points of the nervous centres. It is of such great importance in its diagnostic relations to encephalic lesions that the study of its semiological value may properly engage our attention.

Conjugate Deviation of the Eyes.—Before Adamük discovered in the two anterior tubercula quadrigemina the centres for the movements of the eyeballs, Magendie had established the fact that these movements are under the influence of the middle cerebellar peduncles. This illustrious physiologist, having divided in a hare the middle cerebellar peduncle of one side, saw the corresponding eye turn downward and become more prominent, while the eye of the sound side turned upward and retreated within the orbit. The eyes resumed their normal positions as soon as Flourens divided the middle cerebellar peduncle of the other side. Longet and Schiff, on repeating Flourens's experiment, arrived at a like result. This last observer noticed that when the experimental lesion involved not only the cerebellar peduncle but the lateral region of the cerebellum, the conjugate deviation was still produced, but with an inverse disposition; that is, the eye of the sound side protruded, and was turned downward, while the eye of the injured side became less prominent, and turned upward. A similar fact had already been observed in 1826 by Hertwig.

---

[1] *Gazette hebdomadaire*, No. 33, 1871.

More recently, Curschmann has contended that the conjugate deviation of the eyes, such as had been observed by Flourens, is not the result of section of the cerebellar peduncles, and that this deviation is only produced if the lesion concerns a point in the hemispheres of the cerebellum, which in the hare is known as the acoustic tubercle.

Hitzig, who experimented on rabbits, obtained conjugate deviation of the eyes by applying the two poles of a galvanic pile to the posterior lobe of the vermis. Both eyes deviated to the right or to the left according as the positive pole was applied to the right or the left. When the two electrodes came in contact on the superior lobe of the vermis, one of the eyes turned upward and the other downward, according to the direction of the current.

Finally, Ferrier has seen, when the most anterior part of the vermis was electrically excited, both eyes deviate, that of the right side outward, and that of the left inward. By exciting the middle or inferior part of the vermis, the character of the deviation was reversed. By exciting the cerebellum at various points in the monkey, the dog, and the cat, Ferrier was constantly able to produce conjugate deviation of the eyes, the direction of the deviation varying according to the point excited. In general terms, it may be said that the organs, the excitation of which produces the phenomenon in question, are the tubercula quadrigemina and the cerebellum and its expansions.

Moreover, we see that unilateral lesions of any point whatever of these organs are equally accompanied by conjugate deviation. In fact, if the ocular globes are cut off from the influence of one of the two homologous centres which preside over their associated movements, the influence of the unaffected centre will alone be felt, and the two eyes will take the same anomalous position that would be produced by excitation of a symmetrical point. Clinical experience fully justifies these physiological deductions. For a long time it has been known that lesions situated in the vicinity of the isthmus of the encephalon cause during life hemiplegia, with conjugate deviation of the eyes, accompanied often with a rotation of the head on its axis. In regard to the direction of the deviation, it is variable, sometimes being toward the side on which the lesion is situated, and sometimes toward the opposite side. But this phenomenon of the conjugate deviation of the eyes, with or without rotation of the head, is observed in the cases of lesions affecting very different parts of the cerebral hemispheres. Very often the phenomenon is due to a restricted lesion in the vicinity of the corpus striatum and peduncular expansion. M. Prévost[1] has endeavored to lay down a general law to the effect that, in a case of cerebral lesion, the conjugate deviation of the eyes is always from the affected side. But a case reported by Duplay, and four others by Eichorst, demonstrate that the contrary direction may exist, and that consequently rule enunciated by Prévost is too absolute.

---

[1] "De la deviation conjuguie des yeux." Thèse de Paris, 1868.

IV.

LESIONS OF THE OPTIC TRACTS.

Lateral Hemiopia.—As we have seen, according to the theory generally admitted, the nerve-fibres which form the optic nerves only partially decussate in the chiasma. Those which cross over from one side to the other are, of course, the nearest to the median line, and occupy the most internal part of the nerve and optic tract of each side. On the other hand, those fibres which do not cross occupy the most external part of the nerve and tract. An examination of the drawing (Fig. 33) will be sufficient to make these statements clear, and to establish the fact that the nerve-fibres which form each tract pass to the corresponding half of each retina. Thus the fibres of the left optic tract pass to the left half of each retina, and those of the right to the right half.

FIG. 33.

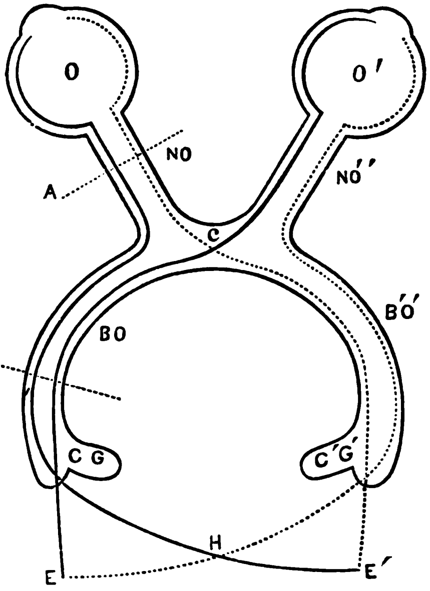


Diagram explaining the phenomena of lateral hemiopia and of crossed amblyopia, according to M. Charcot. ("Leçons sur les localisations cérébrales," 1876, p. 124.) C, semi-decussation in the chiasma; H, decussation posterior to the corpora geniculata; C G, C′ G′, corpora geniculata; O, left eye; O′, right eye; N O, N′ O′, optic nerves; B O, B′ O′, optic tracts; A, lesion of the left optic nerve, producing amaurosis of the corresponding side; B, lesion of the left optic tract, producing right lateral hemiopia; E, lesion producing right crossed amblyopia; C, a lesion at this point will produce temporal hemiopia.

This explanation of the course of the fibres of the optic nerves between the retinæ and the corpora geniculata enables us to perfectly understand the different forms of hemiopia due to lesions of the optic tracts.

Thus *lateral homologous hemiopia*—that is to say, sensorial paralysis of the same half of each retina—has been observed in a great number of cases in which the lesion affected one of the optic tracts either directly or indirectly by the intermediation of neighboring organs. Now the theory given unqualifiedly requires that when a lesion destroys the optic tract of the left side, only the left half of each retina will be deranged in its position, and inversely if the lesion is situated on the right side.

The resulting hemiopia may be confined to one retina when a unilateral lesion only affects the most external fibres (homologous hemiopia) or the most internal (crossed hemiopia) of one of the optic tracts.

When a circumscribed lesion is situated in the anterior angle of the chiasma, so that its action is limited to the most internal of the fibres of the optic nerves, the result should be a *temporal hemiopia*. In other words, in accordance with the theory, the visual trouble should be limited to the internal half of each retina. Soemish has published a case of this kind, in which the diagnosis of the seat of the lesion was made during the life of the patient.

Finally, in order that there should be a *nasal hemiopia*—that is, that the visual trouble should be limited to the external half of each retina—it is necessary that a bilateral and symmetrical lesion should affect only the external part of each optic tract. This has been demonstrated by several cases; among others, by those of Knapp, published in 1873.

V.

LESIONS OF THE CEREBRAL AND CEREBELLAR PEDUNCLES.

a. Cerebral Peduncles.

The cerebral peduncles contain in their substance all the sensory and motor fibres which connect the periphery with the encephalic centres. Two parts, or strata, are to be distinguished in these organs: the one superior, the other inferior. The latter contains exclusively the direct sensory and motor fibres which pass from the gray cortex of the hemispheres to the spinal ganglionic masses without touching the central ganglia of the encephalon. According to Meynert, the sensory fibres occupy the internal part of the peduncles. A slight lesion of these organs will, therefore, produce a complete sensory and motor hemiplegia, but with the peculiarity that the senses of sight and of hearing are in general unaffected. Sometimes the third pair of nerves is affected, and then paralysis of the muscles of the eyeball supplied by it complicates the paralysis of the muscles of the opposite side. [That is, the paralysis of the ocular muscles is on the same side as the lesion, while that of the other muscles is on the opposite side. There is, therefore, to this extent a cross paralysis.—W. A. H.]

A lesion affecting only the external part of a peduncle will produce an exclusively motor hemiplegia; and a lesion limited to the internal portion of a peduncle will cause only hæmianæsthesia.

b. Cerebellar Peduncles.

Curschmann has recently published the case of a tuberculous woman, who complained of vertigo and headache followed by convulsive seizures, at the end of which she fell on the right side. At the autopsy there was found a tuberculous meningitis, and a focus of softening on the right side involving the anterior and posterior cerebellar peduncles. The same author has already proven by experiments that, if a cerebellar peduncle of one side be divided, the animal is at once seized with convulsions, and falls on the side corresponding to the lesion.

More recently Coutry[1] has published the case of a man who presented, as his only symptoms, obstinate vomiting and motor ataxia as regarded certain movements. These latter were jerking and sudden. At the autopsy of this man, who died of tubercular meningitis, there was found entire destruction of the left inferior cerebellar peduncle.

CHAPTER XIX.

SYMPTOMATOLOGY OF CEREBELLAR DISEASES.

THE pathology of the cerebellum is as yet imperfectly understood. This is due, in the first place, to the insufficiency of our knowledge of the functions of this organ, and, in the next, to the fact that very profound lesions may be situated in the cerebellum, and may even destroy it in great part, without causing the least functional disturbance; while circumscribed lesions engender symptoms very variable in character, and due for the most part to compression of contiguous organs.

These symptoms are, moreover, similar to those which result from lesions of the most varied of the nerve-centres. It is only the manner in which they are grouped together, and their greater or less frequency, which enables us to diagnosticate with some degree of assurance that the lesion from which they result is in reality situated in the cerebellum.

---

[1] *Comptes rendus de la Société de Biologie, séance du* 5 Mai, 1877.

Chief among them must be placed—

(*a.*) Headache, vomiting, and vertigo; phenomena that are observed very often in circumscribed lesions of the encephalon, whatever may be their seat, but which are met with more frequently in cases in which they are located in the cerebellum.

(*b.*) Titubation; a symptom which by some physiologists is assigned to derangement of the coördinating faculty which, according to them, resides in the cerebellum.

(*c.*) Motor excitations under the form of epileptic convulsions.

(*d.*) Motor paralyses, which, however, generally consist of feebleness, and not complete loss of power in the limbs.

(*e.*) Sensorial troubles, such as those of sight and hearing.

(*f.*) There are also to be noticed, in the course of cerebellar affections, derangement of the faculty of speech, and paralyses of the face and of certain muscles of the eye; symptoms which are due to the compression of contiguous organs, such as the cerebellar peduncles, the bulb, etc. [To this must be added that peculiar oscillatory motion of the eyeballs, called nystagmus, a phenomenon rarely absent in cases of cerebellar disease.—W. A. H.]

The following table shows at a glance the relative frequency of the several symptoms of cerebellar diseases. It is made up from cases adduced by some among the most eminent of those who have studied the subject:

SYMPTOMS.	Duchek, 15 cases.	Ladano, 56 cases.	Ollivier and Leven, 76 cases.	Luys, 100 cases.
Occipital headache	3	27	31	25
Frontal "	2 } 10	11 } 56	3 } 41	6 } 50
General "	5	18	7	19
Vomiting "	..	..	22	35
Troubles of motility	12	54	71	..
Progressive feebleness of the muscles	4	2	17	45
Uncertainty of gait	4	16	9	28
Hemiplegia	..	7	8	16
Paraplegia	3	2	..	7
Strabismus	3	8	..	..
Facial paralysis	..	3	2	..
Predominance of action on one side of the body	1	..	25	5
Tremor	2	4	..	7
Epileptiform convulsions	2	7	..	12
Derangements of speech	..	..	13	20
Amblyopia—amaurosis	7	23	..	18

Some writers have attached a certain diagnostic importance to the absence in cerebellar diseases of anæsthesia and troubles of general sensibility. Nevertheless, Drozda,[1] of ninety-five cases of cerebellar affections collected by himself, found that in fifteen there were modifications of the general sensibility, not including headache among the number.

---

[1] "Wiener med. Wochenschrift," 1876, p. 155.

On the other hand, the cerebellum having been for a long time considered as the centre for the coördination of movements and of the muscular sense, the presence of motor incoördination and the abolition of the muscular sense have been regarded as the two pathognomonic signs of cerebellar lesions. Clinical observation has demonstrated the incorrectness of this view. Muscular sensibility has been found to remain intact during the existence of lesions of the organ in question, and incoördination is a symptom peculiar to locomotor ataxia, the lesion of which exists in that portion of the spinal cord in which the radicles of the posterior nerve-roots are situated. In the cases of persons suffering from cerebellar disease the gait is vacillating and titubating, like that of a drunken individual; and the symptom is as well-marked when the patient walks with his eyes open as when he has them shut. There is not in these lesions that absence of harmonious muscular action that is observed in ataxics.

Nevertheless, Huppert[1] has quite recently published a case of atrophy of the cerebellum in an individual whose lower extremities were affected very much as are those of patients suffering from locomotor ataxia. To the uncertain and titubating gait are added often motor troubles similar to those which ensue in animals who have been subjected to experimental lesions of the cerebellum and its peduncular expansions. There are irresistible impulsions forward or backward, and a like tendency to turn continually toward the same side; sometimes, also, the patients are unable to stand.

Another characteristic of cerebellar lesions is the rapidity with which, in the great majority of cases, the ultimate phenomena supervene. Contrary to the course of a great number of cerebral lesions, the beginning is insidious, and the fatal termination often supervenes in an unexpected and sudden manner.

On the whole, we are led to diagnosticate a cerebellar lesion when we find united in the same patient a certain number of the following phenomena: Occipital headache with nervous vomiting, vertigo, a staggering and uncertain gait, weakening of the muscular power without ataxia, amblyopia, amaurosis, and an irresistible tendency to turn over toward one side. [In this connection I may be excused for quoting from a paper,[2] to which reference has already been made in other parts of the work, the following conclusions based on original experiments, and which have a bearing upon the subject of cerebellar disease:

"1. The consequences of removal of the cerebellum, if the animal survives the immediate effects of the operation, are not enduring.

---

[1] "Archiv für Psychiatrie," B. vii., 1877, p. 91.

[2] "The Physiology and Pathology of the Cerebellum," *Quarterly Journal of Psychological Medicine*, January, 1869, p. 209.

"2. The entire removal of the cerebellum from some animals does not apparently interfere in the slightest degree, even for a moment, with the regularity and order of their movements.

"3. The disorder of movements, which results in birds and mammals immediately after injury of the cerebellum, is not due to any loss of coördinating power, but is the result of vertigo.

"4. The phenomena of cerebellar disease or injury, as exhibited in man, are not such as show any derangement of the coördinating power.

"5. In those diseases of which the chief phenomena relate to derangement of the coördinating power, the lesion is not in the cerebellum, and the symptoms are altogether different from those due to cerebellar disease or injury."—W. A. H.]

The principal lesions which may affect the cerebellum are tumors, hæmorrhage, softening, and sclerosis, which latter sometimes terminates in atrophy of the organ.

I.

TUMORS OF THE CEREBELLUM.

The cerebellum may be the seat of tumors of very different natures. Thus there are aneurisms or vascular tumors, parasitic tumors (cysticerci echinococci), cancerous tumors, tubercles, syphilitic gummata, sarcomata, lipomata, etc.

It is rarely the case that tumors are situated in the cerebellum without the neighboring parts being more or less affected by compression. Thus it is that the different instances which have been reported present variable groups of symptoms one from the other; and in the future it will be necessary to separate with more exactness than has yet been done the phenomena which are the results of lesions of the cerebellum from those which are due to the compression of contiguous organs.

Ordinarily those patients in whom, on post-mortem examination, a tumor has been found in the cerebellum, have presented, as an initial symptom, pain, generally in the occipital region, accompanied with nausea and obstinate vomiting. Sooner or later come troubles of motility, consisting of titubation, the impossibility of standing erect, a tendency to roll over toward one side or the other, epileptiform convulsions, and general muscular weakness, which does not, however, ordinarily reach the extent of actual paralysis. Statistics show that, in a large number of cases, individuals affected with cerebellar disease have exhibited some form of circumscribed paralysis, either of the hemiplegic or paraplegic form. But such phenomena must, in general, be ascribed to compression exercised on the motor fibres which pass through the cerebral peduncles, the protuberance, and the bulb, by a tumor situated in the vicinity of the isthmus of the encephalon. Derangement of the faculty of speech and tremors of the tongue and lips have also been noticed in a large number of cases of tumor of the cerebellum; and amblyopia, reaching to the point of extreme blindness, is also a very common symptom. M. Raymond[1] has reported a case of a woman, twenty-seven years old, at whose post-mortem examination a tumor was found, the size of a hen's egg, which was situated between the two lobes of the cerebellum, in such a position that it separated one from the other, so that the superior vermis was notably flattened by the pressure. The anterior extremity reached as far as the tubercula quadrigemina, which were also compressed. Relative to the position of the tumor M. Raymond says that it was such that the fourth ventricle was entirely obliterated. It is important, in view of the pathogeny of visual troubles, that the tubercula quadrigemina were entirely destroyed, and that the optic tracts were atrophied—facts which sufficiently account for the amblyopia. M. Raymond has added the abstracts of fifteen other cases of tumor of the cerebellum, accompanied by papillary atrophy with amaurosis.

From the comparison of these different cases, we are warranted in concluding that there is no definite connection between the seat of a tumor in the cerebellum and the development of morbid change in the optic nerves. We know, also, that atrophy of the optic nerves is met with in many other affections, spinal and cerebral, as well as cerebellar. Of sixty cases of tumors of the cerebellum, collected by Macabiau,[2] forty were characterized by troubles of the eyesight. Generally these consisted of a more or less complete amaurosis, the result of atrophy of the optic nerve. In other cases the pupils were dilated or contracted, or there was deviation of the eyeballs to one side or the other.

We therefore perceive that the symptomatology of tumors of the cerebellum is subject to great differences—a fact which renders the diagnosis very difficult; and the fact must not be lost sight of, that a neoplasm of great size may be developed in the cerebellum, and not be manifested by any symptom during the life of the patient, only being brought to light in post-mortem examination.

[A very interesting case of tumor of the cerebellum has recently been published by Prof. Diodato Borrelli, of the Royal University of Naples. In this instance there were numerous sarcomatous growths over the whole surface of the body, and after death similar growths were found in some of the viscera, the spinal cord, the cerebrum, and notably in the cerebellum. The compression exerted by the intracranial tumors had been sufficient to flatten the optic thalami and corpora quadrigemina, and to push these latter out of their normal position.

---

[1] *Gazette Médicale de Paris*, 1871, p. 371.

[2] "Des tumeurs du cervelet," *Thèse de Paris*, 1869.

Two tumors were found in the cerebellum: one, on the left hemisphere, the size of a hen's egg; the other, much smaller, was situated on the periphery of the right lobe.

But it is not so much to the morbid anatomy as to the symptoms that I desire in this connection to call attention. These latter were observed with great care; and though, of course, they were in part due to compression of neighboring organs, they do not differ in this respect from those likely to result in all cases of tumors of any part of the encephalon.

The patient's countenance wore an expression of pain; his eyes were from time to time seized with spasms, during which they turned in all directions; his gait was titubating and uncertain, and he walked with his feet far apart, so as to increase his width of base; there was intense vertigo; the headache was agonizing, and was situated in the frontal and vertical regions, but even more decidedly in the occipital region; vomiting, which was persistent and unamenable to treatment, and a tormenting pruritus, were also present; and there were tonic spasms of the muscles of the neck, by which the head was strongly rotated to the right.

The visual power was diminished, though not equally, in both eyes, and the hearing was similarly affected. The other special senses were not deranged, and the general sensibility of the body was apparently intact. Neither was there any notable impairment of muscular power, though the lower extremities were somewhat more affected in this way than the upper. As to the mental condition, at first sight it appeared as though it were normal, but careful observation showed that the memory was weakened.[1]—W. A. H.]

II.

HÆMORRHAGES OF THE CEREBELLUM.

M. Hillairet, who was the first to study hæmorrhages of the cerebellum by separating them from those occurring in other parts of the body, distinguishes two forms—the one sudden, the other chronic and of slow progress.

In the sudden form the patient is struck with an apoplectic shock, and dies comatose at the end of a short time. In the slow form the intelligence remains intact, the patient complains of headache, generally in the occipital region, and vomiting is very frequent. The other symptoms consist of a vertiginous, titubating gait, and a general weakness of the limbs, difficulties of speech, and troubles of vision. Hemiplegia, which has been considered a common symptom of lesion of the cerebellar substance (Hillairet), should be regarded, according to M. Vulpian, as an effect of the compression exercised by hæmorrhagic centres on the motor fasciculi of the isthmus of the encephalon.

---

[1] Dr. Borrelli follows his account of this interesting case with a very full bibliography of the literature of cerebellar tumors, and a thoroughly well-digested and critical essay upon the subject.

On the whole, the symptoms of cerebellar hæmorrhage are the same as are observed in tumors of this organ, with the exception that their evolution is much more rapid. In fact, even in the slow form of the disorder, it is rare that the patient survives more than a few days.

III.

ATROPHY OF THE CEREBELLUM.

Atrophy of the cerebellum, when it is not congenital, is generally consecutive to sclerosis of that organ. It can not be recognized by any positive symptom. Sometimes it is met with in epileptics. M. Duguet has reported three examples of this condition. In a case of very pronounced atrophy of the cerebellum, in a young man twenty-two years of age, Max Huppert[1] has noticed during life epileptiform convulsions, choreiform agitation of the muscles, with diminution of muscular force, difficulty of standing erect, titubation during walking, and trouble of speech. The limbs, which were not paralyzed, were nevertheless affected with incoördination similar to the same symptom as observed in ataxics.

A case of what is probably sclerosis and atrophy of the cerebellum was for several years under my observation, and was presented by me before the American Neurological Association at its meeting in June, 1877.[2]

The patient, a boy about four years of age, was brought to my clinique at the University Medical College in January, 1876. He was apparently in good health, was well grown for his age, and had not been subject to any exhausting disease. As he sat upon a chair, he exhibited no indications of paralysis, spasm, or incoördination. He moved both legs well and with normal force, and could use either hand in the ordinary way. But it was impossible for him to assume the erect posture, and when he attempted to do so he stood in a peculiar, one-sided, stooping position, the left arm being strongly flexed against the side of the chest, while the right was thrown out behind him. He could not maintain himself on his feet without support.

---

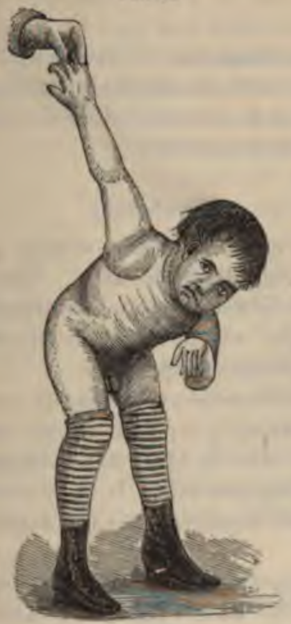
[1] "Archiv für Psychiatrie und Nervenkrankheiten," B. iii., p. 98.

[2] "On a Hitherto Undescribed Form of Muscular Incoördination," "Transactions of the American Neurological Association, 1877."

The attitude is shown in the accompanying woodcut, Fig. 34, taken from a photograph.

In walking he was able to direct his steps with a certain amount of precision, but yet not to a normal extent. He appeared also to have more difficulty in arresting his movements, and was accordingly apt to come up violently against obstacles which were in his way. His gait was rather a run than a walk, and he often fell. In bringing the case before the class I expressed the provisional opinion that it was one of chorea paralytica, but further examination, and the inefficacy of all treatment, soon caused me to change this view.

FIG. 34.



In May he came under the charge of a surgeon who circumcised him, under the impression that the case was one of reflex incoördination. It is scarcely necessary to say that the operation was unsuccessful. When he appeared before the Association, in June, there had been a gradual advance in the intensity of his symptoms. Yet, notwithstanding the marked incoördination, there was no paralysis, no derangement of sensibility, no bladder disturbance, no spasm, no diminution of electric excitability of the muscles, and none of the peculiar symptoms indicative of sclerosis of any part of the cord.

After this there was a short intermission in his symptoms, and his father thought he was recovering. He wrote me to that effect, September 7th, no medicine having been taken. But soon afterward he again relapsed, and his condition gradually became worse. When I last saw him, about a year ago, there were nystagmus and a total inability to stand. When he tried to do so, he bent over till his head touched the floor, and thus he remained, apparently endeavoring to stand on his head. When he wished to go anywhere in the room, he lay down on the floor and rolled toward it, turning over toward the left always. About this time Dr. J. S. Jewell, of Chicago, saw the patient. Continued examination and study of this very interesting case lead me to the opinion that it is one of sclerosis and atrophy of the cerebellum.

The subject of the diagnosis of diseases of the brain cannot be passed over without a reference to the masterly work of Nothnagel[1] on the subject, even if that reference does not go further than citing the conclusions at which he arrives:

Cerebellum.

"1. Diseases of the cerebellum may remain completely latent, and thus be incapable of being diagnosticated. This is generally the case with destructive lesions situated in one hemisphere.

"2. Lesions of slight extent may, on the other hand, present a very variable and complicated appearance.

"3. The most characteristic symptoms of cerebellar affections are incoördination, a titubating gait, and intense vertigo. These symptoms are, however, met with in other brain-diseases, and cannot, therefore, be considered pathognomonic. It is by a consideration of *all* the phenomena, positive and negative, that the diagnosis of cerebellar diseases is to be made.

"4. Cerebellar staggering always denotes the involvement of the middle lobe, whether this be the primary situation of the lesion or an implication (functional also) through pressure.

"5. At the same time, incoördination and vertigo may be absent in diseases of the cerebellum situated mainly in the hemispheres, as is also the case sometimes with tumors the seat of which is the vermis. If in such a case we feel justified on other grounds in suspecting the existence of a lesion in the posterior encephalic region below the tentorium, we can never, with any approach to certainty, diagnosticate the existence of either primary or secondary diseases of the cerebellum. Its implication under such circumstances is possible, but is by no means a matter of certainty.

"6. In addition to the symptoms given under section 3, there are some others which can be considered as indicating the existence of a disturbance of the functions of the cerebellum, and hence the presence of lesions of this organ. Perhaps certain derangements of speech (anarthia) in cases of extensive atrophy of the cerebellum may be so regarded, but yet there is no surety on the subject.

---

[1] "Topische diagnostik der Gehirnkrankheiten; eine klinische Studie," Berlin, 1879.

"7. Vomiting, when constant and severe, may in some cases support the diagnosis of a cerebellar disease, but it is not conclusive of such a condition, for it is often an accompaniment of other encephalic affections. It is lacking in all cases of destructive lesions, and is not a constancy in those due to pressure from diseased contiguous organs.

"8. The like is true also of amblyopia and amaurosis, and of choked disk and optic neuro-retinitis.

"9. Headache is likewise only met with in cases of pressure from diseased contiguous organs. Its fixed situation in the posterior cranial region may in some cases indicate the existence of a cerebellar disease, but it is no more pathognomonic of such an affection than its presence in the frontal region would indicate a healthy cerebellum.

"10. The most diverse derangements of the motor and sensory cerebral and spinal nerves may exist in conjunction with cerebellar disease, but only in cases of lesions due to pressure. They are not, therefore, in instances of disease of the cerebellum, of any diagnostic importance. On the contrary, they are very apt to lead to errors of diagnosis. Still, however, if we can, out of all the symptoms, select some one of derangement of motor or sensory nerves, we may find important indications toward the exact localization of the lesion. Thus, for instance, paralysis of the whole of the right facial nerve indicates the existence of a tumor on the corresponding side, and decided hemiplegia its seat on the basilar surface. Generally, however, we must be careful not to draw definite conclusions in this respect.

"11. Psychical derangements are absent. Only under the general conditions which exist in all brain-lesions, whatever their situation, are we apt to meet with them in affections of the cerebellum. Nevertheless, they are probably ordinary phenomena in general atrophy of the organ."

Crura Cerebelli.

"1. Stationary destroying lesions of the crus-cerebelli, producing complete destruction of the same, cause no characteristic symptoms of diagnostic value.

"2. Irritative lesions alone produce such symptoms, and then only when the connection of the crus with the cerebellum is not interfered with. Hæmorrhages only produce symptoms in the beginning.

"3. These symptoms consist in forced positions of the trunk, head, and eyes, rotatory motions on the long axis of the body, and in vertigo, with the inclination to fall to one side.

"4. Of the foregoing symptoms, the only ones which are characteristic are the position of the eyes and rotatory movements of the body observed by Nonat, as all the rest, so far as they have been clinically noticed, have been recognized as being due also to other localized lesions.

"5. On the contrary, the turning of the body, together with a like movement of the head and eyes, indicates the existence of a crus-cerebellar lesion.

"6. The direction of these movements is sometimes toward the healthy side and sometimes toward the diseased side, but as yet the cause of these differences is not known.

"7. Whether or not disturbances of coördination and ataxia result from lesions of the crus-cerebelli, is not yet ascertained.

"8. The foregoing remarks refer entirely to the median crus to the pons. There is nothing to be said relative to the anterior and posterior crura."

Pons.

"1. Stationary intrapontine destructive lesions derange in their functions the motor, sensory, and vaso-motor nerves of the extremities, and the fifth, sixth, seventh, eighth (?), eleventh (?), and twelfth cerebral nerves. Pressure-lesions may cause derangement of function in the ninth and tenth cerebral nerves.

"2. The number of nerves involved varies greatly in individual cases—according to the size and exact seat of the lesion. Still, we cannot at present determine with any accuracy, from the implication of any one nerve, which part (in cross-section) of the pons is the seat of the lesion.

"3. In many cases non-progressive lesions of the pons produce the same group of symptoms as others located in the cerebrum, and cannot be distinguished from them.

"4. Difficulties of articulation, and even inability to speak, are more frequent in cases of lesions of the pons than in other localized affections, except in those of the medulla oblongata. These, when existing in conjunction with the group of symptoms referred to under section 3, indicate with some degree of probability a lesion of the pons.

"5. The whole group of diseases of the pons, destructive lesions as well as tumors, have an individual character when accompanied by cross-paralysis. This, when present, is a most important diagnostic sign. Nevertheless, it is not pathognomonic of affections of the pons, as it may also occur in the course of basilar affections. In the latter case, however, we have slowly-advancing disorders, such as tumors and chronic meningitis. The quick and sudden onset of a well-marked form of cross-paralysis is a pretty certain indication of the existence of an intrapontine lesion.

"6. This cross-paralysis involves the motor and sensory nerves of the extremities on the side opposite to that of the lesion, and the trigeminus abducens, facial (auditory ?), and hypoglossus, on the side corresponding to that of the lesion. Within this range, the form of the cross-paralysis may vary greatly in individual cases.

"7. The motor and sensory (and vaso-motor ?) paralysis of the extremities is always on the side opposite to that of the lesion. The cerebral nerves mentioned in section 6 are sometimes affected on the opposite side, and sometimes on the same side as the lesion. The paralysis of the facial is on the same side.

"8. Whether or not the conjugate ocular paralysis of the external rectus muscle of the same side and the internal rectus of the opposite side is an indication of the existence of a lesion of the pons, is still an open question.

"9. It is true that anæsthesia in lesions of the pons is in proportion somewhat more frequent than in lesions of the cerebrum. Still, this fact is of no importance as a diagnostic sign. So far as relates to the implication of special nerves, it may be said that, if the abducens be paralyzed, and the other symptoms indicate at the same time the existence of an intracerebral lesion, this will almost certainly be located in the pons.

"10. Difficulty of swallowing is of no great importance as a diagnostic mark. It may sometimes assist in the formation of an opinion, but cannot be the foundation for one.

"11. The same is true as regards disturbances of respiration and of the circulation.

"12. Ataxia is seldom found in affections of the pons, but when present is not contraindicative of their existence. Still, it is so rarely met with, so very rarely in comparison with its frequency in cerebellar lesions, that it cannot take a high position as a diagnostic mark.

"13. Other motor phenomena, such as compulsory movements, a tendency to go backward, swinging movements of the extremities, and the like, are in the present state of knowledge more curious than valuable as diagnostic marks. Lying on one side, and lateral movements of various kinds, have not yet been observed in man unless with the implication of the crus-cerebelli.

"14. Spastic phenomena of individual muscles are seldom encountered, and are only of importance as aids to diagnosis when accompanied by particular groups of symptoms (trismus, for instance).

"15. General epileptiform convulsions are only of diagnostic value in cases of hæmorrhage and embolism. They are not present in stationary lesions and in tumors.

"16. Symptoms connected with sensation are not probable in cases of affections of the pons. As regards the auditory nerve, the number of observations is as yet too few to enable us to arrive at definite conclusions. Nevertheless, loss of hearing on one side must be considered as of some importance in forming a diagnosis. Contracted pupils occurring in the course of an apoplectic attack point to the pons as the probable seat of the lesion.

"17. Vomiting, headache, and vertigo, occur in pressure-lesions, under the same conditions as when they are the result of disease of other parts of the brain."

Medulla Oblongata.

"1. The diagnosis of lesions of the medulla oblongata cannot be made with any degree of certainty. Generally there is not even a suspicion of such a localization, as the only phenomenon due to such a lesion is paralysis of the extremities. Tumors appear sometimes to be entirely latent.

"2. The most important and characteristic symptoms of lesion of the bulb are those due to implication of nerve-roots. These consist of dysæsthesia, anæsthesia, dysphagia, aphonia, and disturbances of respiration and of the circulation. On the other hand, destroying lesions much less frequently produce disturbances of the inferior facial branch than is the case in chronic progressive bulbar paralysis.

"3. Of the above-named phenomena, aphonia and respiratory and circulatory disturbances are of much more importance as aids to diagnosis, since they are not observed as symptoms of destroying lesions of other parts of the brain.

"4. If any one of the symptoms mentioned under section 2 be wanting, it aids us in locating more exactly the seat of the lesion. It also helps us to distinguish the lesion from that producing the complicated condition known as progressive bulbar paralysis.

"5. The extremities are very often involved, and generally in the form of motor paralysis, either as hemiplegia or paraplegia. Well-marked anæsthesias have not as yet been observed.

"6. But in the presence of an apparently typical case of 'apoplectiform bulbar paralysis' it is necessary to be cautious in forming a diagnosis, as a quite analogous group of symptoms may, although seldom, be developed as the consequence of lesion of the cerebrum. For the distinction between the two, perhaps the existence of reflex actions, and absence of aphonia and circulatory and respiratory disturbances in the latter, must be taken into consideration."

Cerebral Peduncles.

"1. That lesions of the cerebral peduncles give rise to well-marked symptoms, cannot be said with absolute certainty.

"2. Motor, sensory, and vaso-motor tracts may be involved by lesions of the cerebral peduncles. Whether all these, or one or other of them, be affected at the same time, depends on the extent and situation of the lesion.

"3. The paralysis is manifested in the nerve-tracts of the extremities of the opposite side (very seldom there alone), and in the facial, hypoglossal, and trigeminus, also, of the opposite side.

"4. The phenomena mentioned under sections 2 and 3 afford no diagnostic marks of disease of the crura cerebri, as they are also manifested in diseases of the upper part of the pons or of the corpus striatum.

"5. The characteristic symptom is a paralysis of the oculo-motor nerve of the same side, in conjunction with paralysis of the nerves of the upper and lower extremities, or facial, of the opposite side [cross-paralysis]. This alternate paralysis may exist in conjunction with basal morbid processes. A lesion of the peduncle may only with confidence be suspected when they occur suddenly and at the same time. Tumors may occasionally cause paralysis of the opposite oculo-motor.

"6. Motor and sensory disturbances of single nerve-tracts occur only as consequences of tumors, and are of no importance as characteristic marks for diagnosis of the exact seat of the lesion.

"7. Abnormalities in the excretion of urine are of no diagnostic value."

Tubercula Quadrigemina.

"1. The diagnosis of lesions of the tubercula quadrigemina is very difficult and uncertain, for the reason that the symptoms are sometimes exceedingly ambiguous and at others scarcely noticeable.

"2. From a consideration of the results of our present experience it appears that the symptoms due to lesion of the anterior and posterior pair respectively are different. The implication of the anterior pair (a few cases are in opposition) is accompanied with diminution of the sense of sight, or even blindness. This symptom is, however, of very ambiguous character, and must not be necessarily referred to lesion of the tubercula quadrigemina unless it is of sudden development and accompanied with choked disk, optic neuritis, and optic atrophy; for, although these changes are due to tumors of these organs, they also occur from tumors of other parts of the brain. The diagnostic value of amaurosis appears to us as of most importance under the following circumstances: when it is sudden and acute, with non-mobility of the pupils, accompanied with other symptoms of lesion of the brain and with negative ophthalmoscopic results.

"4. Lesions of the posterior pair are accompanied, but not always, with paralysis or paresis of the oculo-motor nerve. Its existence is not, however, any more than its absence, an infallible mark for diagnosis.

"5. Great importance is to be attached to the character and appearance of the oculo-motor disturbance—a unilateral paralysis may exist from a bilateral lesion, and, when unaccompanied with alternate paralysis of the extremities, points to the tubercula quadrigemina as the organs involved.

"6. This bilateral implication of the motores oculorum appears to be sometimes due to a unilateral lesion of the tubercula quadrigemina.

"7. Relative to the state of the pupil nothing exact is known. It appears, however, that lesion of the anterior pair arrests its reactions.

"8. From lesions of the posterior pair it appears that disturbances of equilibrium and coördination may result, similar to those due to cerebellar disorders."

Thalami Optici.

"1. Relative to the majority of the symptoms regarded as being due to lesions of the thalamus, it is very doubtful if they have a direct relation to this organ, or only occur indirectly through implication of neighboring cerebral parts. Other manifestations, really the result of thalamic lesion, are of uncertain import, or they occur also from disease of other encephalic organs.

"2. From which it follows that a certain diagnosis of an isolated thalamic lesion is, in the present state of our knowledge, in the majority of cases impossible. Only under a very favorable combination of circumstances is it possible to arrive at a definite conclusion.

"3. Motor paralysis cannot, in our opinion, support the idea of a possible thalamic lesion. On the contrary, when paralysis exists we must suppose other parts to be involved, even if the thalamus is the principal seat of the lesion.

"4. The like is true of sensory paralysis. If, through the relations which exist between injuries of the part of the internal capsule near the thalamus, and sensibility, it is sometimes concluded that the lesion is situated near the thalamus or in it (in such a manner that the internal capsule is also implicated), we even then are not warranted in diagnosticating the existence of a thalamic lesion.

"5. That which is said under section 4 is true also of vaso-motor tracts.

"6. Disturbances of sight may occur through lesion of the posterior third of the thalamus; whether, however, this be crossed amblyopia or homomymous hemiopia, cannot yet be determined with certainty. But such visual disturbances do not indicate with any degree of sureness the existence of thalamic lesions, as they may occur with other localized brain-diseases, such as those of the occipital lobes, corpora quadrigemina, and optic tracts.

"7. A peculiar series of irritative motor disturbances, such as hemichorea, athetosis, and unilateral tremor, are possibly due to thalamic lesion. Nevertheless, if this fact were definitely established, these phenomena would, even in undoubted cases of thalamic disease, be of little value diagnostically, as they may occur when the lesion has a very different location.

"8. That a diminution or increase of reflex excitability indicates disease of the thalamus is incorrect.

"9. Possibly, disturbances of the muscular sense, and—

"10. Disorders of psycho-motor reflex actions, are indications of thalamic lesions. Further observations and investigations are, however, necessary on these points.

"Taking everything into consideration, in the present state of our knowledge, a lesion of the optic thalamus may, perhaps, under a particularly favorable combination of circumstances, be diagnosticated, if the conditions stated under sections 6, 7, 9, and 10, be present, but even then this cannot be done with certainty."

Corpora Striata.

"1. Destroying lesions of the corpus striatum may produce crossed motor, sensory, and vaso-motor paralyses.

"2. If the lesion be not too small, motor hemiplegia is uniformly present.

"3. This hemiplegia may gradually disappear if the lenticular or caudate nucleus be alone involved. It remains, however, if the internal capsule be involved, whether alone or in conjunction with the gray nuclei. In these permanent paralyses—that is to say, those due to lesions of the internal capsule—there is often subsequently secondary muscular contraction.

"4. The motor hemiplegia resulting from stationary destructive lesions affects constantly both extremities of one side and the inferior branch of the facial nerve. Usually the muscles of the trunk are also rendered paretic. The hypoglossal is either not at all or only in the beginning affected. Its implication is seldom permanent.

"In rare cases the extremities or the facial are alone involved.

"5. The symptoms of lesion of the lenticular nucleus are not of such a character as to admit of their distinction from those due to lesion of the caudate nucleus.

"6. Motor paralysis is the sole symptom, if the lesion is situated only in the anterior third of the corpus striatum in the region supplied by the lenticular striated artery.

"7. In some cases hæmianæsthesia is an accompaniment of the hemiplegia. This is characterized by the fact that along with the cutaneous anæsthesia the nerves of special sense—sight, hearing, taste, and smell—on the corresponding side are affected; still, these latter are not necessary features in hæmianæsthesia due to lesions of the corpus striatum, as the condition is generally confined to the skin.

"8. The hæmianæsthesia shows the implication of the most posterior part of the internal capsule with the contiguous part of the corona radiata; still, lesions may exist in the posterior part of the internal capsule between the optic thalamus and the lenticular nucleus without the production of anæsthesia.

"9. Generally the hemiplegia and the hæmianæsthesia exist together. It is only occasionally that the first disappears and that th latter remains.

"10. Occasionally disturbances in the functions of vaso-moto innervation—increased temperature, redness, etc.—occur in the paralyzed parts. These indicate the implication of the posterior portio of the internal capsule.

"11. Hemichorea often occurs in conjunction with hæmianæsth sia. Nevertheless, its relations to the corpus striatum cannot at pre ent be determined with accuracy."

Centrum Ovale.

In regard to lesions of the white substance of the brain—constituting the centrum, no very definite conclusions are reached by Nothnagel. Thus stationary destroying lesions of occipital, spheroidal, and anterior and middle frontal portions, give rise to no well-marked symptoms. The same is true of pressure-lesions.

The most important symptom is that resulting from lesions of the anterior and posterior central regions—motor paralysis of the opposite side, like that caused by lesions of the corpus striatum or cortex.

Aphasia is a probable symptom of lesion of the white substance of the foot of the third frontal convolution.

The Cortex.

"1. Disease of the surface of the brain—that is, of the gray substance and the immediately contiguous white substance—produces in one group of cases decided symptoms, in another they are without symptoms, remaining latent.

"2. Psychical derangements indicate, in general, disease of the superficies of the brain (Hirnoberfläche), but exact localization cannot as yet be made.

"3. Dysphasic and aphasic symptoms indicate disease of the cortex."

This assertion is not absolutely correct, for lesions without direct implication of the cortex may, under certain conditions, occasionally produce aphasia (as, for example, of the centrum ovale). Nevertheless, such cases are so rare, and the proportion is so small, that for all purposes of clinical diagnosis the proposition under section 3 may be accepted.

"4. In simple ataxic aphasia the third left frontal convolution is the first locality to be thought of, and next the island of Reil.

"This proposition is of course true also for amnesic or ataxi-amnesic disturbances of speech.

"5. If, as some observations seem to show, affections of other parts of the brain may cause aphasia, this is so seldom the case that the location assigned under section 4 should always be the first brought to mind. Besides, it is impossible, from the character of the disturbance of speech, to determine the exact seat of the lesion.

"6. Word-deafness indicates with great probability a lesion of the left parietal lobe, especially of the first temporal convolution.

"7. Hemiopia does not of itself indicate the existence of a cortical lesion. At best we can only suspect such, and then probably in the occipital lobe, when the condition in question is developed suddenly, as a single symptom, and with entirely negative ophthalmoscopical phenomena—perhaps after an apoplectic attack.

"8. Unilateral disturbances of vision may occur as the consequence of lesions of the superficies of the brain. Hitherto they have only been observed with diffused cortical lesions, such as progressive paralysis and cysticerci. As to their importance in locative diagnosis, nothing of any positiveness can be said.

"9. Sensory disturbances of the skin have as yet no importance in the diagnosis of diseases of the cortex.

"10. With great hesitation we make the suggestion that unilateral disturbance of the muscular sense, when it appears by itself without attendant phenomena, perhaps indicates the existence of a lesion of the parietal lobe.

"11. Motor derangements occur in conjunction with cortical lesions, and under certain circumstances may by their character enlighten us in regard to the latter.

"12. Sometimes the paralysis appears as a simple hemiplegia, such as is ordinarily the result of a lesion of the corpus striatum, with or without secondary contractions in the paralyzed extremities. In this case the diagnosis is impossible. Nevertheless, the belief that a cortical lesion existed would be strengthened if there were also aphasia. But even then the motor paralysis might be due to a lesion of the third frontal convolution coexisting with one of the corpus striatum.

"If, in connection with paralysis of the extremities and the facial and the hypoglossal nerves, there is ptosis, it is probable that a cortical lesion exists.

"On the contrary, decided disturbances of sensibility occurring in conjunction with motor hemiplegia indicate either that the lesion is not cortical, or that, if it is, it is extensive, and extends deeply into the white substance of the centrum ovale.

"13. Relatively, paralyses due to lesions of the cortex are often monoplegias, partial hemiplegias, isolated paralyses of the facial, the hypoglossal, and the nerves of the arm (rarely of the leg), or of the arm and leg, or arm and face.

"14. These monoplegias, their intracerebral origin being first determined, indicate, not with absolute certainty but with great probability, a cortical lesion.

"15. The character and development of these monoplegias alone are of no consequence as indicating a cortical lesion.

"16. On the other hand, certain kinds of motor irritative phenomena are of great value in the diagnosis of cortical lesions.

"17. These appear sometimes as partial convulsions of individual muscles, and occur either as the consequences of hæmorrhage or softening, or by the development of a tumor, and which subsequently are followed by paralysis of the affected muscles. In such cases we may with great probability, but not with certainty, suspect a lesion of the cortex.

"Or partial cleonic convulsions make their appearance in the already paralyzed region. In such cases, judging by our present experience, a lesion of the cortex exists.

"18. In other cases the motor phenomena are those of a general epileptiform attack, and with the peculiarity that the typical recurring spasm always begins in the same group of muscles in an extremity or half of the face. This form of convulsion is always developed after an existent paralysis. It may be regarded as a probable symptom of lesion of the cortex.

"19. The existence of motor symptoms from lesion of the cortex indicates that the seat of the morbid cause is in the anterior central and posterior central convolutions and the paracentral lobule."

SECTION II.

DISEASES OF THE SPINAL CORD.

CHAPTER I.

SPINAL CONGESTION.

THOUGH congestion of the spinal cord, like that of the brain, is of two kinds, active and passive, yet the symptoms and general course of the two varieties are so generally alike, that nothing would be gained by considering them separately.

Symptoms.—The symptoms of spinal congestion are referable to the cord and to those parts of the body below the seat of the lesion. The most prominent local phenomenon is pain, which is rarely acute, but is described as a dull, aching sensation similar to that experienced in the back after severe and long-continued muscular exertion in a stooping attitude. This pain is increased by the recumbent posture and by standing, if the lower part of the cord be its seat; but pressure, if steadily applied, does not augment it. A sudden blow or a shock, such as that produced by making a false step, aggravates it to a considerable extent.

A sensation of heat is occasionally experienced in the cord, which is not unpleasant, and which is not affected by pressure.

With the local symptoms there are others still more notable perceived in the parts of the body below the seat of the disease. Thus, if as is very generally the case, the lesion be situated in the dorsal or lumbar region, there are disturbances of sensibility and motility in the lower extremities. The various sensations indicating anæsthesia are present, and are usually first experienced in the skin covering the under surface of the toes. Formication, "pins and needles," tingling, and a feeling as if the toes are swollen, are noticed. It is rarely the case that the anæsthesia is complete. Its extent and exact situation may be accurately determined by the æsthesiometer.

Sometimes there is hyperæsthesia, and occasionally both conditions coexist. The extent of either may be definitely measured with the æsthesiometer. Shooting pains in the limbs and along the course of the nerves coming from the diseased part of the cord are now and then present, but they are not a prominent feature in simple congestion.

A sensation of constriction is at times complained of, and is referred to the body or one or both of the limbs. It is compared to the feeling which would be produced by a tight cord, or encasement in an unyielding garment. It is rare in uncomplicated spinal congestion. According to the situation of the lesion, there are pains either in the abdomen, chest, or both, and there may be dyspnœa and palpitation of the heart. In three cases under my care, the difficulty of breathing and irregular cardiac action were prominent features. Similar cases are cited by Ollivier[1] (d'Angers). The temperature of the parts of the body below the lesion is always reduced, from the fact that the vaso-motor nerves are involved.

Erections of the penis are common, especially after the patient has been in the recumbent position for some time.

The most striking phenomena of spinal congestion are those connected with the alterations of motility. Paraplegia is always present to some extent, though it is rarely complete. Thus the patient, though unable to walk, can generally move the legs when sitting down or lying in bed. Twitchings of the muscles are occasionally present, but not often to a severe degree.

The loss in the power of motion, like the alterations in sensibility, is only present in those parts of the body situated below the diseased parts of the cord. The bladder is very generally affected, either in its own muscular tissue or in its sphincter. In the first case, there is a difficulty of expelling the urine, owing to loss of expulsive power, and this is aggravated by paralysis of the abdominal muscles, or there is incontinence of urine from paralysis of the sphincter. Both conditions may coexist, and then, when a sufficient quantity of urine has accumulated in the bladder, it dribbles away. In such a condition, the bladder is never entirely empty, and the urine is passed alkaline and fetid.

The sphincter of the rectum is sometimes involved, producing involuntary evacuation of the fæces, but obstinate constipation from paralysis of the abdominal muscles, and consequent loss of expulsive power, are much more common. Reflex excitability is, according to my experience, invariably lessened, and is sometimes entirely abolished.

The electro-muscular contractility of the paralyzed muscles is always more or less diminished, though not to the same extent as in some other affections of the cord. As a general rule, the farther the muscle is from the centre the less is its electro-muscular contractility.

---

[1] "Traité des maladies de la moelle épinière," troisième édition, Paris, 1837, tome iii., pp. 1–137.

The tendency of spinal congestion is to extend itself and eventually to involve the whole cord. In the active form of the disease, this process often takes place with great rapidity, and the symptoms generally are more pronounced and succeed each other with more promptness. The phenomena of spinal congestion are always rendered more decided by the patient's assuming the recumbent posture. He is hence more paralyzed in the morning before rising from bed than in the evening before he retires. This is due to the fact that the position in question, especially if he lies on his back, allows the spinal blood-vessels to become more readily distended. It is the same thing as regards the cord, that keeping the head in a dependent position would be as regards the brain.

Bed-sores are not common. Radcliffe[1] seems to assert that they are never met with. Brown-Séquard[2] says an ulceration upon the sacrum or nates is not rare in this affection. Ollivier[3] does not mention them in his account of the disease. Of the large number of cases of spinal congestion that have come under my observation, bed-sores occurred in but two, and in these there was reason to believe they were not the special result of the lesion of the cord.

According as the antero-lateral or posterior columns are mainly affected, the symptoms of spinal congestion differ. Thus, in the former case, the phenomena are chiefly manifested as regards motility, in the latter as regards sensibility. Generally both sets of columns are involved. In spinal anæmia, as we shall presently see, this is not the case.

Causes.—The most common cause of spinal congestion, according to my experience, is exposure to intense cold. Fevers appear to be next in frequency, especially those of malarious origin, and the excessive use of alcoholic liquors probably comes next as a causative influence.

Venereal excesses, and maintaining the erect posture for a long time, were the obvious cause in several cases. This last influence was very well marked in the case of an eminent lawyer of this city, who became suddenly affected with spinal congestion after making a speech of several hours' duration. The suppression of a customary discharge, such as the menstrual flow or a hæmorrhoidal bleeding, is likewise liable to induce congestion of the cord. I have recently treated two cases, in which the congestion of the cord followed the cold stage of intermittent fever; and it is occasionally the result of blows and falls. I have seen several cases which were due to railway injuries.

In one of these the patient, an elderly gentleman, was violently thrown to the floor of a railway-car in consequence of a collision with a stationary train in front. At first, there were nausea, vomiting, slow and feeble pulse, and very great nervous prostration—in fact, all the more prominent symptoms of shock. The injured man revived after the administration of stimulants, and was partly enabled to walk to an hotel near by. On the following day, however, pain was experienced in the lower dorsal region, and difficulty was experienced in moving the legs. Sensibility was impaired in the lower extremities. The bladder was partially paralyzed, and in consequence the urine was drawn off with the catheter. From this time his condition became worse, till at last, sensibility was entirely abolished in both lower extremities, and the power of motion was altogether lost. The bladder never became entirely paralyzed. The sphincters both of the bladder and rectum remained unaffected. When I saw him, three months after the reception of the injury, he was incapable of feeling the prick of a pin in any part of his body below the first lumbar vertebra, and could not move a single muscle of the lower extremities. There were no bedsores, and the condition of the limbs was not impaired. Electro-muscular contractility, though greatly lessened, was not completely lost. A two-cell galvano-faradic battery, when used with its full power, failed to produce any contraction; but all the muscles responded feebly to the interrupted primary current from a hundred-cell battery. Reflex excitability was entirely lost. There were no twitchings or spasmodic contractions of the paralyzed muscles. Nor had any such movements ever been noticed. Under the use of ergot, iodide of potassium, and the primary galvanic current to the spine and the muscles of the lower extremities, he recovered so far as to be able to walk short distances with crutches, and obtained full control of his bladder, but there was no marked improvement in the sensibility.

---

[1] Lectures on the "Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities," Philadelphia, 1861, p. 69.

[2] *Op. cit.* [3] Reynolds's "System of Medicine," vol. ii., p. 622.

In another case, the affection was apparently induced by excessive muscular exertion. The patient went to bed, feeling fatigued, and in the morning was entirely paraplegic. The paralysis gradually extended upward, till on the third day both arms were devoid of power. On the fifth day I saw him. He was then deprived of voluntary power in both upper and lower extremities. Reflex excitability was notably impaired, as was also the electric contractility of the muscles. He, however, had full power of the bladder and sphincters. On the sixth day the left side of his face became paralyzed. He was treated as the case just described, and recovered entirely in the course of about two months. He has remained perfectly well ever since, and able to attend to his business as a commercial traveler.

Leudet[1] reports several cases in which symptoms similar to those present in the foregoing examples were produced by falls and excessive muscular exertion, and which were, in his opinion, instances of spin

---

[1] "Sur la congestion de la moëlle survenant à la suite de chûtes et d'efforts violen *Archives généraves*, 1860, tome i., p. 257.

congestion. In a subsequent paper[1] he returns to the subject and adduces additional cases in support of his view. In his opinion the congestion, with its accompanying symptoms, is produced at the end of a period often of some hours after the operation of the exciting cause. Sometimes the congestion is limited, affecting only a segment of the spinal cord; at others it, from the very first, involves the cord throughout its entire length. This fact explains the circumstance that the symptoms are by no means uniform, either as regards their character or location.

Among the effects of working under compressed air, spinal congestion must be included. Drs. Babington and Cuthbert,[2] of Dublin, have called attention to this fact; and Dr. Clark,[3] of St. Louis, has recently brought forward several additional cases occurring in the workmen in the caisson used in building the bridge over the Mississippi River.

Passive spinal congestion may be caused by any obstruction to the return of blood by the veins, such as cirrhosis of the liver, pregnancy, abdominal tumors of various kinds, diseases of the lungs or right side of the heart, and the long-continued maintenance of the dorsal decubitus.

Diagnosis.—Spinal congestion is liable to be confounded with several other affections, and with some to the great injury of the patient. Thus it may not be distinguished from spinal anæmia, a condition likewise giving rise to paraplegia, but of which the treatment is very different.

It may be diagnosticated from anæmia of the posterior columns by the facts that in it there is pain in the cord, increased by pressure on the spinous processes of the vertebræ, or, if there is no spontaneous pain, such pressure causes it; by the disturbance induced in the cranial, thoracic, or abdominal viscera, according to the part of the cord affected being much more prominent; by the circumstance that women are more generally its subjects; and that, when there is paralysis, it is hysterical and transitory in character. In anæmia of the antero-lateral columns there is often a previous affection generally of the urinary organs which has caused the anæmia, or some other source of reflex irritation or exhaustion can be discovered. Besides, in spinal anæmia, either of the posterior or antero-lateral columns, the symptoms are less strongly marked after the patient has been lying down some time, whereas the reverse is the case in congestion.

Spinal anæmia never produces any urinary derangement, although such trouble may cause spinal anæmia. In a case, therefore, in which there was doubt as to the spinal cord being in a state of congestion or anæmia, the order of sequence, as regards the paraplegia and bladder-difficulty, would seem to render the diagnosis exact. In spinal anæmia the bladder is affected before the paraplegia appears; in spinal congestion the paraplegia comes on before the bladder is involved.

---

[1] "Recherches cliniques sur la congestion de la moëlle à la suite de chûtes ou d'efforts," Clinique médicale de l'Hôtel-Dieu de Rouen, Paris, 1874.

[2] "Paralysis caused by working under Compressed Air," *Dublin Quarterly Journal of Medical Science.*

[3] *St. Louis Medical and Surgical Journal.*

In spinal anæmia there is no formication, pricking, tingling, or other sensation indicative of anæsthesia. Hyperæsthesia is, on the contrary, exceedingly common.

The further diagnostic marks will be considered when we come to the subject of spinal anæmia.

Congestion is distinguished from inflammation of the cord by the facts that in it the jerkings of the limbs are slight, that the paralysis is not so extreme, that the urine is never alkaline, unless there is paralysis of the bladder, that the pain in the cord is less, and by the infrequency of the feeling of constriction at the upper limit of the lesion.

From meningitis it is diagnosticated by the absence of spasms in the muscles of the back, and by the fact that movements of the paralyzed limbs do not cause pain.

Prognosis.—In simple uncomplicated spinal congestion the prognosis is not unfavorable, if, in addition, the case be put under suitable treatment at an early period. It must be remembered, however, that there is a tendency to interstitial changes, and that, if the vessels of the cord be left for a long time in a state of turgidity, it may be impossible to prevent structural alterations of greater severity. In some cases, especially those of traumatic cause, the symptoms are quite evanescent, disappearing in the course of a few hours. It is better, therefore, for the physician to be guarded in expressing his prognosis in such instances when recent, till sufficient time has elapsed for the tendency of the morbid process to be manifested.

Morbid Anatomy.—The post-mortem appearances in cases of congestion of the spinal cord are either in the cord proper or its membranes. As regards the first, section shows increased vascularity both of the gray and the white substance, especially if microscopical examination be made. The capillaries will be found increased in size and more numerous than in the normal condition.

The membranes of the cord contain very large and very tortuous vessels, and in congestion they are rendered still larger and more complex in their anastomoses. The pressure which they are capable of exerting upon the cord is not inconsiderable.

It is almost invariably found that the cerebro-spinal fluid is increased in quantity.

These evidences of congestion are sometimes extremely limited in their extent, at others the whole length of the cord is involved.

Pathology.—The symptoms which result from congestion of the cord are of two distinct classes: increased excitability from hyperæmia, and interruption of the proper functions of the cord from pressure.

The former, in the main, results from the increased amount of blood in the gray matter and white substance; the latter from the enlarged meningeal vessels and the increased amount of cerebro-spinal fluid, which, in the form of serous effusion, is the result of their turgidity. As one or the other of these conditions predominates, we have some symptoms more prominent than others. Thus hyperæsthesia indicates rather hyperæmia of the gray substance, anæsthesia pressure upon the white substance. Twitchings, when present, are likewise the result of over-excitation of the gray tissue; while motor paralysis is induced by pressure upon the antero-lateral columns.

The modifications which may be produced in the intensity of the symptoms by the position of the body show the effect of pressure very clearly. In the recumbent posture on the back, the blood gravitates in large amount to the spinal vessels, pressure on the cord is increased, and the phenomena of anæsthesia and paralysis are more strongly marked. Again, causes which increase the activity of the circulation, such as alcoholic stimulants, and others which directly augment the amount of blood in the cord, such as strychnia and phosphorus, invariably increase the hyperæsthesia and induce muscular twitchings, even if they have not previously been observed.

Treatment.—In cases of spinal congestion which come on suddenly, and which are therefore acute in their character, such as result from the sudden arrest of an habitual discharge, sudden and violent muscular exertion, or falls, blood may be drawn locally from the spinal region by cups or leeches. The best place for the application of the latter is the verge of the anus, and I have several times witnessed very decidedly satisfactory results from their use in this situation.

Purgatives are likewise beneficial, and preference should be given to those which produce watery evacuations, as thereby the overloaded vessels are relieved, and the absorption of the superabundant cerebro-spinal fluid facilitated. Nothing can be better for this purpose than the sulphate of magnesia given in doses of a drachm two or three times a day.

In this form the ergot of rye may be given with advantage from the very inception of the disorder. In the more chronic form it is indispensable. It should be administered in very much larger doses than are laid down in the text-books on materia medica. I am in the habit of using it in this and analogous spinal diseases, in doses of a drachm of the fluid extract three times a day. The action of the ergot is to lessen the diameter of the blood-vessels of the cord by its constringing power over the organic muscular fibre entering into the composition of their walls. Ten years ago[1] I spoke as follows: "But I have recently ascertained by actual experiment that ergot does not exert the influence in question. I prepared a weak aqueous infusion of this substance and placed it on the web of a frog's foot under the microscope. In a few moments contraction of the capillaries ensued, and they became so small as not to allow of the passage of the blood-corpuscles. This experiment I have repeated several times, and am perfectly satisfied that the result is as I have stated. More, I have frequently injected small quantities of the infusion into the stomach of frogs, and contraction of the capillaries of the web always followed."

---

[1] "A Clinical Lecture on Chronic Myelitis, delivered in the Baltimore Infirmary," March 16, 1681, *American Medical Times*, June 15, 1861, p. 379.

These experiments, therefore, fully confirmed those made a short time previously by Dr. Brown-Séquard.

Since that time I have given it in a large number of cases of diseases of the spinal cord, congestion among them, in which it was necessary to diminish the amount of blood in the spinal vessels, and I am entirely satisfied that such is its effect; but I never obtained its full influence till, in accordance with the suggestion of Dr. A. Jacobi, of this city, I adopted the practice of giving it in what may be called very large doses. Among the cases which first came under my care, since my residence in New York, was that of Mr. W., of Tennessee, who had become affected with congestion of the cord, from exposure to cold and dampness. When I first saw him he was unable to walk without the assistance of crutches, and a man on each side of him holding his shoulder. He had paralysis of the bladder, which had come on after the paraplegia, and a constant, dull, aching pain in the loins. There were also occasional startings of the legs, especially after he had gone to bed. All his symptoms were worse in the morning. I at first gave him ten drops of the fluid extract of ergot three times a day, but, continuing this for two weeks without effect, I at once increased the doses to a teaspoonful. In less than a week the effects were manifest. Sensibility began to return in the extremities, the strength increased, the bladder began to contract on its contents, the lumbar pains ceased, and by the end of a month he had entirely recovered. A few weeks afterward he had a relapse, but the ergot, taken as before for ten days, again restored him, and he has since remained perfectly well.

In the case of Mr. T., of Norfolk, Virginia, whose affection was apparently the result of exposure to cold and dampness, and who was barely able to walk with two canes, a complete cure was accomplished by the use of ergot continued for about a month. In two other cases occurring in mechanics of this city, ergot was the only remedy employed, and both were entirely cured in less than a month.

In several cases I have administered the ergot hypodermically in doses of five grains daily of Beaujon's extract, but I am convinced that nothing is gained by this course. As regards the efficacy of ergot in spinal congestion, there is not, in my opinion, any doubt. Even when it fails to effect a cure, its good influence is at least shown for a time. I would as soon think of treating intermittent fever without quinine as congestion of the spinal cord without ergot.

Belladonna is also a valuable remedy in spinal congestion, especially when there is paralysis of the sphincter, or when the pain in the back is severe. The tincture, in doses of fifteen drops three times a day, may be employed, and a belladonna plaster may be applied to the painful region of the spine.

The hot douche—the water being of the temperature of 98° Fahr.—to the spinal column is an excellent means of determining the blood from the deep to the superficial vessels. The water should be allowed to fall from the height of about two feet upon the naked back over the diseased part of the cord every day for about five minutes. Dry cups are also valuable adjuncts.

Electricity is always useful. The constant current should be applied to the spine over the affected part of the cord, and the intensity and quantity should be as great as the patient can endure without much discomfort. I am not sure that it makes any difference in which direction the current be passed. Of its benefit I have no doubt. The duration of the application should not exceed ten minutes. The beneficial effect is probably due to the diminution of the calibre of the blood-vessels through its action on the vaso-motor nerves.

The induced current should be used to the paralyzed muscles, so as to excite them to contract. In this way their nutrition is promoted, and any tendency to atrophy from disuse obviated.

The primary current should not be employed more frequently than every alternate day. The induced may be used every day for half an hour or longer, short of causing fatigue.

I will only add that strychnia and phosphorus should never be administered in congestion of the cord, as their action is the very reverse of that desired, and irreparable damage may be done by their use.

CHAPTER II.

SPINAL ANÆMIA—ANÆMIA OF THE POSTERIOR COLUMNS.—ANÆMIA OF THE ANTERO-LATERAL COLUMNS.

A DEFICIENT quantity of blood in the spinal cord, or a depravation in the quality of the blood circulating through its tissue, gives rise to two cognate, but, so far as their phenomena go, different affections. In one of these, which has hitherto been known as spinal irritation, the morbid action is in a great measure confined to the posterior columns of the cord; in the other, which embraces several differently-named disorders, characterized by paralysis, such as reflex paralysis, inhibitory paralysis, spinal paresis, paralysis from peripheral irritation, etc., the antero-lateral columns are mainly affected.

In thus specifically locating the lesions in these affections, I am aware of the fact that post-mortem examinations are wanting to support them. Nevertheless, the symptoms characteristic of each are so distinctly marked, and are in such intimate physiological relation with the regions of the cord specified, that I do not think I am at all exceeding the limits of probability.

Retaining the name of spinal irritation, as one well known to the profession, it will nevertheless be understood that, in my opinion, the proper designation of the disease would be anæmia of the posterior columns of the spinal cord. I have arrived at this view after a very careful consideration and analysis of the symptoms observed in a large number of cases.

The same remarks are applicable, *mutatis mutandis*, to reflex paraplegia, a symptom which I am very sure results from anæmia of the antero-lateral columns of the cord.

ANÆMIA OF THE POSTERIOR COLUMNS OF THE SPINAL CORD.—SPINAL IRRITATION.

History.—It has been questioned by several distinguished authors whether such an affection as spinal irritation really exists as a distinct disease. Thus Valleix[1] ascribes the most important of its manifestations to hysteria, and regards the spinal tenderness present as being due to simple intercostal neuralgia; Inman[2] considers the pain produced by pressure over the spinous processes of the vertebræ as existing in the muscular attachments, and as indicative of what he calls myalgia. Mr. Skey[3] evidently looks upon all cases of spinal irritation as hysterical in their character, and Niemeyer[4] speaks incredulously on the subject, without giving any very decided opinion. It would be easy to bring forward other authorities who have expressed similar views, and I may have to allude to some of them more fully hereafter. In the recently-published nomenclature of the Royal College of Physicians,[5] the affection has no place unless it be included under the head of hysteria.

The first author who distinctly grouped together the symptoms of spinal irritation was J. Frank,[1] who, under the name of rachialgia, described the disorder with considerable accuracy, and laid the principal stress upon the local pain. He was followed by Stiebel,[2] who, however, contributed little to our knowledge of the subject.

---

[1] "Traité des névralgies, ou affections douloureuses des nerfs," Paris, 1841, p. 345.

[2] "On Myalgia: its Nature, Causes, and Treatment," etc., second edition, London, 1860, p. 225, *et seq.*

[3] "Hysteria," etc., New York, 1867, p. 72, *et seq.*

[4] "A Text-Book of Practical Medicine," American edition, New York, 1869, vol. ii. p. 258.

[5] "The Nomenclature of Diseases drawn up by a Joint Committee appointed by the Royal College of Physicians of London," London, 1869.

Mr. J. R. Player[3] was among the first English physicians, if not the very first, to call attention to the fact that eccentric derangement of function may be the result of irritation of the spinal cord. Thus he says: "Most medical practitioners who have attended to the subject of spinal disease must have observed that its symptoms frequently resemble various and dissimilar maladies, and that commonly the function of every organ is impaired whose nerves originate near the seat of disorder. The occurrence of pain in *distant parts* forcibly attracted my attention, and induced frequent examination of the spinal column; and, after some years' attention, I considered myself enabled to state that, in a great number of diseases, morbid symptoms may be discovered about the origins of the nerves which proceed to the affected parts, or of those spinal branches which unite them; and that, if the spine be examined, more or less pain will commonly be felt by the patient on the application of pressure about or between those vertebræ from which such nerves emerge."

The term "spinal irritation" appears to have been first used by Dr. C. Brown,[4] of Glasgow, who, in a very excellent paper, gives a picture of the disorder which cannot fail to be recognized as truthful and exact by those who have witnessed several cases of the affection. He insists upon not confounding the complaint with those organic diseases of the vertebræ and spinal cord which some of its symptoms cause it to resemble, points out the variation of the phenomena according to the seat of the spinal tenderness, and inculcates the employment of rest and counter-irritation as the most effectual remedies. His ideas of the pathology of the disease are: "That the immediate cause of the pain of the back and breast is spasm of one or other of the muscles arranged along the spine altering the position of the vertebræ, or otherwise compressing them as they issue from the spinal marrow.

"That this spasm in many instances is strictly a *local* disease, produced by fatigue, wrong posture, or other causes, and quite unconnected with the state of the brain, spinal marrow, or nervous system in general.

"But that, in other formidable instances, this partial, spasmodic, or wrong action of the muscles, is owing to a faulty state, perhaps an enlargement, of the vessels of the brain or spinal marrow. This state of the brain, as in many other diseases, gives rise to spasm or even to convulsion of certain muscles; which partial symptom, from its severity, attracts the chief attention. This local affection is confined to those portions of the spine where there is the greatest motion, and where, of course, the muscles having the greatest activity are most liable to deranged action or spasm. I imagine that this view of the subject is illustrated and perhaps confirmed by various symptoms which were observed in the different cases, and which without it were very incomprehensible. The partial palsy, the affection of the sight, the giddiness of the head (for I find that this was a prominent symptom in several cases, especially in that of A. S.), all give some confirmation to the notion that the brain is affected in these severe cases."

---

[1] "De Rachialgitide" in Prax. med. univ., P. II., t. i., p. 37.

[2] "Ueber Neuralgica Rachitica," *Rust's Magazine*, t. i., c. xvi., p. 549.

[3] *Quarterly Journal of Science*, vol. xii., p. 428. Quoted by Teale.

[4] "On Irritation of the Spinal Nerves," *Glasgow Medical Journal*, No. II., May, 1828.

Dr. Darwall,[1] of Birmingham, describes several features of the affection with accuracy, such as those simulating cardiac and gastric diseases. He is inclined to believe that the morbid condition of the spinal cord depends mainly upon irregularity of the circulation, generally congestion.

But no essay upon the subject of spinal irritation, which had yet appeared, was equal in thoroughness to that of Mr. Teale,[2] and it is to him that the views now generally held relative to the connection between various eccentric phenomena, such as pain, spasm, and visceral disturbance, and a peculiar condition of the spinal cord, are to be attributed. He, however, committed the great error of regarding the affection as being due to inflammation, and, in what for those days was logical accordance with this theory, he combated it with strong antiphlogistic measures. His book may be studied with advantage, as presenting an admirable account of the many diverse phases which spinal irritation may assume.

Mr. Tate,[3] in his work on hysteria, attributes many of the protean manifestations of this disorder to spinal irritation, limited, however, to the dorsal region. He fails to recognize it as an independent disease. His treatment consists in the application of tartar-emetic ointment along the whole length of the dorsal vertebræ, and strong purgation. He discountenances the use of leeches and blisters.

Mr. W. R. Whatton[4] insists chiefly upon the liability to mistake spinal irritation for disease of the vertebræ. He gives a very excellent account of the symptoms. The treatment he recommends consists in the abstraction of blood, by leeches or cups, from the parts where the tenderness is felt, repeated every three or four days, and the application of small blisters on each side of the painful spots. Any debility ensuing in consequence of this treatment is to be remedied by the preparations of iron and quinine.

---

[1] "On some Forms of Cerebral and Spinal Irritation," *Midland Medical Reporter*, May, 1829.

[2] "A Treatise on Neuralgic Diseases dependent upon Irritation of the Spinal Marrow and Ganglia of the Sympathetic Nerve," London, 1829.

[3] "Treatise on Hysteria," London, 1830.

[4] "On Spinal and Spino-Ganglial Irritation," *North of England Medical and Surgical Journal*, No. III., 1831.

In a clinical lecture delivered in Dublin, Dr. Corrigan[1] relates the particulars of several cases of spinal irritation, successfully treated by local antiphlogistic measures, and the internal use of iron. He does not, however, add any thing of importance to our previous knowledge of the subject.

Dr. Isaac Parish,[2] of Philadelphia, appears to have been the first American author who called attention to the affection in question. He relates the details of several cases, recommends the use of counter-irritants, especially tartar-emetic ointment, and concludes:

"First, that tenderness on pressure in some portion of the spinal cord is an attendant on many chronic neuralgic affections, and that, by relieving it in the manner proposed, these complaints are either entirely eradicated or temporarily suspended.

"And, secondly, that the precise indications which this circumstance affords are not sufficiently understood at the present time to justify the establishment of any definite pathological principles applicable to the whole class of neuroses."

Dr. W. Griffin and his brother, Mr. D. Griffin,[3] of Limerick, were the next to write upon the subject. The joint work of these gentlemen is based upon one hundred and forty-eight cases, all of which are thoroughly analyzed, and from which very definite deductions of pathology and treatment are drawn. The essay is not excelled in importance by any previous contribution, and constitutes a really valuable study. The conclusions which they draw are so instructive that I do not hesitate (though by no means indorsing them all) to transfer them without abbreviation:

"1. That tenderness at one or more points of the spine is an attendant on almost all hysterical complaints, on numerous cases of functional disorder when the hysteric disposition is not so obvious, and in many nervous or neuralgic affections.

"2. That many of the symptoms of these affections evidently depend upon a peculiar state of certain nerves, probably at their origin, may be reproduced at any moment by pressure, and are often relieved by remedies applied there.

"3. That, in all cases of tenderness of the cervical and upper dorsal spine, there was nausea, or vomiting, or pain of stomach, or affections of the upper extremities; but no pain of the abdomen, dysury, ischury, hysteralgia, or affections of the lower extremities.

---

[1] *Medico-Chirurgical Review*, July, 1831, p. 182.

[2] "Remarks on Spinal Irritation as connected with Nervous Diseases: with Cases," *American Journal of the Medical Sciences*, vol. x., 1832, p. 223.

[4] "Observations on the Functional Affections of the Spinal Cord and Ganglionic Nerves, in which their Identity with Sympathetic, Nervous, and Simulated Diseases is Illustrated," London, 1834.

"4. That, in all cases of dorsal tenderness, pains affecting the abdomen, bladder, uterus, testes, or lower extremities, were usual symptoms; while nausea, vomiting, or affections of the upper extremities, were never complained of.

"5. That nausea and vomiting appeared to have more relation to tenderness of the cervical spine, pain of stomach to tenderness of dorsal; but that, when there was soreness of both, nausea or vomiting was still more frequent, and pain of the stomach scarcely ever absent.

"6. That, when several points or a great extent of the spinal column is painful and tender on pressure, local remedies are generally less effectual, and there is a strong disposition to transference of the disordered action from one organ to another; the pain or tenderness in all such cases of transference, shifting its place to a corresponding part of the spinal column, leaving the original point free, or with a very diminished degree of tenderness.

"7. That spinal tenderness is seldom or never met with in cases of pure inflammation, except when these accidentally occur in persons previously suffering from irritation of the cord; and that, when appearances of inflammation present themselves in any organ accompanied by a corresponding spinal tenderness, they cannot commonly be removed by the remedies applicable to inflammatory cases, and are often rendered worse by them.

"8. That there does not appear to be a complaint to which the human frame is liable, whether inflammatory or otherwise, which may not be occasionally irritated in disturbed states of the cord; and hence that this disturbed state is one vast source of those complaints called hysterical or nervous.

"9. That those functional disorders connected with spinal tenderness are very often attended by some disturbance of the functions of the uterus, but that they are by no means always so, since they occur in those who are regular in this respect: in girls long before the menstrual period of life, in women after it has passed, and, lastly, in men of nervous susceptible habits, and in boys.

"10. That in fact they are not necessarily dependent upon any one organ; since they are found indifferently coexisting with disturbance of the digestive organs solely, or the uterus solely, or of the circulatory or respiratory system.

"11. That from the cases detailed we have reason to suppose spinal tenderness may arise from uterine disorder, from dyspepsia, from worms in the alimentary passages, from affections of the liver, from mental emotions, from the poison of typhus, from marsh miasmata, from erysipelatous, rheumatic, and eruptive fevers, and from the irritation arising from local injury.

"12. That it is almost invariably found, in connection with gastric or abdominal tenderness, in fever; and this tenderness is, probably, like the soreness of scalp, pains in the limbs, etc., dependent on the morbid state of the cord.

"13. That, whether in fever or in other complaints, it is met with in the situation of the eighth or ninth dorsal vertebra much more frequently than at any other part of the spine.

"14. That affections attended by spinal tenderness are seldom fatal; that, even in those cases of intense irritation of the cord under which patients suffer extremity of pain for years, the event is generally favorable.

"15. That they frequently, as well as hysteria, occur with all the appearances of a primary affection of the nervous system.

"16. That affections are occasionally met with presenting all the marks of the hysteric character, and perfectly resembling cases described as those of spinal irritation, but unattended by spinal tenderness or any other direct indication of a morbid state of the cord."

The treatment recommended consists in the removal of the cause if this still continues in action, purgatives, the application of blisters and leeches to the skin, the internal administration of hyoscyamus and belladonna, to lessen the nervous irritability, alum in cases of gastric derangement, and change of air and scene.

In a subsequent work, the Messrs. Griffin[1] again discuss the subject, but bring forward no additional facts.

Dr. John Marshall[2] is confident that many visceral affections, such as heart-diseases, asthma, phthisis, dyspepsia, diabetes, chorea, and even phlegmasia dolens, are frequently really produced or simulated by spinal irritation. Some of his cases of supposed functional disorder of the spinal cord are, however, obviously organic, consisting of congestion, inflammation, or softening of the organ.

In his classical work, Ollivier[3] devotes considerable space to what he calls "*an Affection described under the name of Spinal Irritation.*" He considers the pathological condition to be one of congestion of the meninges of the cord, and bases this opinion in great part on the success which, according to him, ensues on the use of leeches, blisters, and counter-irritant ointments. In addition, he favors the administration of opium, digitalis, hyoscyamus, belladonna, and subcarbonate of iron.

Türck[4] regards the phenomena of spinal irritation as being due, first, to disorder of other organs, whereby a morbid impression is propagated along the incident excitor nerves to the spinal cord; or, second, to derangement of the capillary circulation of the cord. That is, the disease may be either of eccentric or centric origin. He does not advance our knowledge beyond the point reached by previous authors.

---

[1] "Medical and Physiological Problems: being chiefly Researches for Correct Principles of Treatment in Disputed Points of Medical Practice," London, 1845.

[2] "Practical Observations on Diseases of the Heart, Lungs, Stomach, Liver, etc., occasioned by Spinal Irritation, and on the Nervous System in General as a Source of Organic Disease," London, 1835.

[3] "Traité des maladies de la moelle epinière," troisième édition, Paris, 1837, tome seconde, p. 209.

[4] "Abhandlung über spinal Irritation," u. s. w., Wien, 1843.

Coming again to our own country, we find that in 1844 a very valuable paper was published by Prof. Austin Flint,[1] based upon fifty-eight cases of functional disorder connected with an abnormal condition of the spinal cord. In this memoir, without going into any discussion relative to the pathology of the affection, Dr. Flint considers the disorder as giving rise to tenderness over the vertebral column, causing alterations of sensibility, as affecting the muscular system, as producing abnormal mental manifestations, as affecting the digestive organs, the genito-urinary organs, the heart and circulation, and as causing paroxysms of sinking. He then considers the physical habits of the patients, the results of medical treatment, the probable remote causes, and then, at some length, the remedial measures which he has found most successful. Under this head, Dr. Flint advises the use of counter-irritants to the spine, especially cupping, and generally without scarification. Issues he found inapplicable, death ensuing in the one case in which he used them. There is no doubt, however, that in this instance he had an organic disease to deal with, and that the issues had nothing to do with the fatal result. Tonics, especially iron, he found to be of great advantage.

In a very full analysis of the medical reports of the Stockholm Hospital by Dr. Magnus Huss,[2] the subject of spinal irritation receives due consideration. Dr. Huss classes the symptoms of the disorder as follows: 1. Pain of various parts of the vertebral column, existing either idiopathically or developed by pressure. 2. Cramps, either of a clonic or tonic nature, in those parts subjected to the influence of the spinal cord. 3. Loss of power in the same portions of the body, ranging from simple stiffness and weakness to complete paralysis. 4. Altered sensibility, either by excess or by great diminution of sensation.

It will be observed that in this enumeration the author confines his specification of morbid phenomena to those which relate to sensation and the power of motion.

The treatment is fully and philosophically considered. Of external remedies he prefers counter-irritants, using the milder forms first, and then the severer, such as the moxa and the actual cautery, should the first fail. Venesection, either general or local, should be cautiously employed, and is not generally indicated. He is the first, so far as my researches extend, to mention electricity, a means which he thinks may be employed with advantage in chronic and debilitated cases. Potash-baths are also recommended.

---

[1] "Observations on the Pathological Relations of the Medulla Spinalis," *American Journal of the Medical Sciences*, April, 1844, p. 269.

[2] *British and Foreign Medical Review*, October, 1846, p. 463.

Of internal remedies he specifies iron, opium, strychnia, phosphorus, and valerian, as being preëminently useful.

Axenfeld[1] devotes a considerable portion of his treatise to spinal irritation. He regards it as being produced either by a trouble of innervation or congestion. In the treatment, leeches occupy the first place, and in light cases blisters, sinapisms, dry cups, and stimulating frictions, are useful. Internally he recommends nothing but quinine and iron.

Dr. Radcliffe[2] writes very sensibly on the subject of spinal irritation, and gives a typical case which is quite instructive. He incidentally gives it as his opinion, that the pathological condition is one of anæmia, and he consequently discourages the use of leeches, relying mainly on blisters and tonics.

Leyden[3] declines to recognize spinal irritation as a distinct pathological entity, regarding it as a condition which may result from other primary affections. In this he very generally mistakes cause for effect. His remarks are evidently more based on theory than practice, for it is very apparent he has seen little or nothing of the disorder under consideration.

Rosenthal[4] barely mentions it under the head of hysteria.

Erichsen,[5] with more practical acumen, says of spinal anæmia, and especially of anæmia of the posterior columns of the cord, that it is "a condition which we rather recognize clinically than pathologically, by analogy than by direct post-mortem demonstration, by therapeutical rather than by physiological tests. But yet it is a condition which is now fully recognized as probable, in lieu of positive evidence, by the best and most modern writers on nervous diseases, and one the probable existence of which we may accept."

I have thus cited the principal authorities upon spinal irritation, without, however, by any means, exhausting the bibliography of the subject. Notwithstanding the eminence of many of those who have contended for the existence of a definite affection of the spinal cord, characterized by tenderness on pressure over one or more of the vertebræ, and certain eccentric disorders involving sensibility, the power of motion, and functional derangement of many of the viscera, it must be confessed that the great mass of the medical profession has regarded the whole theory with suspicion, if not with absolute distrust. The principal reason for this is undoubtedly to be found in the fact that, like many other new theories, that of spinal irritation has been applied to explain conditions which it could not logically be made to cover. Thus many cases of disease or disorder of the heart, due to organic difficulties of that organ, or excited by disease of other viscera through the sympathetic system, have been attributed to spinal irritation. The same is true also of the uterus, stomach, liver, and other organs, and even of the spinal cord itself, which often, when the seat of organic diseases, such as congestion, meningitis, inflammation, tumors, etc., has been regarded as simply in a state of irritation. It is very certain, also, that numberless cases of hysteria have been attributed to irritation of the spinal cord. In the following remarks I will endeavor to be as explicit as possible, and not to claim too much for a pathological condition which I am very sure exists, and which I therefore think is entitled to recognition. If I contribute any additional information, it will be mainly due to the fact that our means of examination are much more perfect and extensive, and our knowledge of physiology, pathology, and therapeutics, more thorough than when most of the authors I have quoted wrote upon the subject. My observations are based upon a careful study of one hundred and twenty-seven cases which have occurred in my private practice during the last six years, and of which I have full notes, and twenty-nine cases of which I have less complete data—in all, one hundred and fifty-six cases.[1]

---

[1] "Des Névroses," Paris, 1863, p. 284.

[2] Reynolds's "System of Medicine," London, 1868, vol. ii., p. 640.

[3] "Klinik der Rückenmarks-Krankheiten," zweiter Band, erste Abtheilung, Berlin, 1875, p. 1, *et seq.*

[4] "Klinik der Nerven-Krankheiten," Stuttgart, 1875, p. 440.

[5] "On Concussion of the Spine," etc., London, 1875, p. 188, *et seq.*

Symptoms.—CENTRIC SYMPTOMS.—1. *Tenderness at some one or more Points over the Spinal Column, increased by Pressure.*—This is the essential symptom of spinal irritation, though varying in intensity from the slight degree of pain experienced upon strong pressure to the acute hyperæsthesia which does not allow of even the contact of the clothing without the production of great suffering. It is generally complained of by the patient, though occasionally it has to be sought for by the physician. The brothers Griffin found this symptom present in all but five out of one hundred and forty-eight cases, and it is very probable that these five were not cases of spinal irritation, a supposition which the authors themselves evidently entertain. Certainly the details of the cases do not support the view which would ascribe their phenomena to any affection of the spinal cord. Most of the other authors I have cited refer to this tenderness as a prominent feature. Parish thinks it alone is to be relied upon as indicating irritation; Mr. Whatton declares that it is never wanting; Axenfeld regards it as the dominant and characteristic symptom; and Radcliffe, while admitting that it is not equally well marked in every case, states the rule to be that spinal tenderness and spinal irritation go together.

---

[1] Since the first edition of this work was published, a large additional number of cases of spinal congestion have come under my notice, but, as I have kept no full record of them, I have allowed the statement in the text to remain unaltered.

On the other hand, Flint does not regard tenderness as an invariable and essential element of the affection under consideration. He found it absent or indistinct in five of his fifty-eight cases, while the other attendant circumstances furnished unequivocal evidence that the diagnosis was correct.

My own opinion would lead me to consider no case as one of spinal irritation in which tenderness on pressure over the vertebræ was absent. In the one hundred and fifty-six cases noted by me, this symptom was present in all. There are diseases of the spinal cord, which produce derangements of other organs of the body, and which are not characterized by vertebral tenderness, but these are far more serious affections than spinal irritation, and of altogether different pathology.

The seat of the tenderness is generally in the dorsal region of the spine. The Griffins found cervical tenderness in twenty-three cases, cervical and dorsal tenderness in forty-six, dorsal alone in twenty-three, dorsal and lumbar in fifteen, lumbar in thirteen, the whole spine tender in twenty-three, and no tenderness in five. Of one hundred and forty-eight cases, therefore, one hundred and seven exhibited tenderness in the dorsal region.

Dr. Flint found cervical and dorsal tenderness in three cases, lumbar and dorsal in ten, and dorsal alone in twenty-one cases.

Of my own cases, twenty-five had cervical tenderness only, thirty-seven cervical and dorsal, forty-five dorsal only, nineteen dorsal and lumbar, fifteen lumbar only, and in fifteen the whole spine was tender. One hundred and sixteen cases, therefore, of one hundred and fifty-six were characterized by dorsal tenderness, and in forty-five it was limited to this region.

The degree and character of the tenderness are subject to great variation. In some cases strong pressure is required to develop it, while in others the least touch is insupportable. Sometimes there are shooting pains, which radiate from the tender spot, while at others the hyperæsthesia is quite circumscribed. In a gentleman now under my care with well-marked spinal irritation, and who has a tender spot over the third lumbar vertebra, pressure not only causes intense suffering at that point, but develops pain along the whole course of the crural nerves and their branches as far as their terminations on the inner sides of the feet. Another, a lady, who has spinal tenderness over the eighth cervical and first dorsal vertebræ, experiences, from pressure, intense pain along the course of the first intercostal, the internal anterior thoracic, and all the nerves of the left upper extremity. Why in these and other cases particular nerves should be affected, is a question which will be more fully considered hereafter.

The pain developed by pressure is not always of the same character. Sometimes it is dull and aching, and at others sharp and lancinating. I have not noticed that any very definite relation exists between the character of the pain and the severity of the other symptoms, though as regards the degree of pain of each kind, there is a marked connection. By this I mean that a dull, aching sensation may indicate as profound a pathological condition, and be accompanied by as intense eccentric phenomena, as a sharp and lancinating pain, though a severe aching pain and a severe lancinating pain always indicate more serious disorder than when these sensations are not so emphatic.

The character of the pain varies in accordance with the tissue which it is felt. The dull aching sensation is only developed by strong pressure, and is seated in the muscular, tendinous, or cartilaginou structures about the vertebræ. The sharp, piercing twinges excited by slight pressure arise from the skin, and subcutaneous cellular tissue. With these species of sensations, the æsthesiometer always shows increased sensibility of the skin over and in the vicinity of the painful centres.

To ascertain whether or not the tissues outside of the spinal canal are in a state of hyperæsthesia, the pressure should be applied with gradually-increasing force, by means of the thumbs applied to the spinous processes and the intervertebral spaces, as recommended by Flint. The examination should be thorough, and extend throughout the whole extent of the vertebral column. The fact that the patient denies the existence of tenderness should have no weight with the physician. Only a few days ago a young lady consulted me for severe infra-mammary pain, headache, and nausea. I at once suspected spinal irritation, but she declared, in answer to my inquiries, that there was no sign of tenderness anywhere over the spinal column. I insisted, however, on a manual examination, and to her great surprise found three spots that were exceedingly painful to slight pressure. This young lady had been treated for dyspepsia for several years, without deriving any benefit from the measures used, but was cured by the treatment which I shall presently fully consider. Occasionally it happens that the tenderness is not perceived for some time after the pressure is made. In a recent case I found the interval to be over a minute, and then acute pain, following the course of the nerves, was experienced. I am not prepared to offer an explanation of this phenomenon.

2. *Pain in the Spinal Cord.*—The tenderness just noticed is seated primarily externally to the vertebral canal, and is developed by pressure. That which is now to be considered is located in the spinal cord, and is, therefore, capable of being produced by pressure upon non-tender spots. It is a very common symptom, having been present in one hundred and one of my cases. Generally it is confounded with spinal tenderness, from which, however, it is quite distinct. It is aggravated by motion of the spinal column, by action of the muscles which have their attachments to the spinous and transverse processes, by percu

sion, and sometimes by the erect posture. In the case of a gentleman of this city, it was so great when he stood up that he was forced to keep the recumbent position nearly the whole time. When I first saw him he was wearing an apparatus designed to keep the weight of the head from the vertebral column, and to prevent the vertebræ pressing upon each other, under the idea that he had disease of the intervertebral substance. I removed the instrument, and, treating him for spinal irritation, he recovered his health in a few weeks.

Pain in the spinal cord, in the disorder under consideration, is usually seated near the point of external tenderness, though it is often at a distance, and sometimes is felt throughout the whole extent of the cord. The eccentric phenomena bear a distinct anatomical and physiological relation to it, as do those which are connected with spinal tenderness. There is likewise a similar connection existing between the pain in the cord and the vertebral tenderness.

To ascertain the existence of spinal pain, when it is not spontaneously felt or superinduced by muscular exertion, percussion should be practised. The ends of the fingers will answer for this purpose, though I prefer a little vulcanized India-rubber hammer, and a plessimeter, such as are sometimes used for percussing the chest. Even over spots which exhibit much tenderness, the deep-seated pain in the cord itself can clearly be distinguished.

ECCENTRIC SYMPTOMS.—By far the most important and noticeable symptoms of spinal irritation are to be found in distant parts of the body. These vary in their character and seat, according to the part of the spinal cord affected. Following the example of the Griffins, I shall consider these symptoms as they depend upon irritation of the several regions of the cord with which they are connected.

a. The Cervical Region.—Of the cases upon which this chapter is based, in twenty-five the irritation existed in the cervical region only, of the spinal cord; in thirty-seven, the cervical tenderness was conjoined with dorsal tenderness, and in fifteen with tenderness of the whole spine. Taking the uncomplicated cases as presenting the clearest features, the following would appear to be the more prominent symptoms of cervical spinal irritation.

Vertigo was an accompaniment in eleven cases, and *headache* in fifteen; *noises in the ears* in eight, and *disturbances of vision* in four. *Fullness* and a *sense of constriction* across the forehead were complained of in several cases, as was also tenderness of the scalp. In addition, the *mind* was more or less affected in every case, and in seven the aberration was of such a character as almost to amount to insanity. In one of these, a married lady, aged thirty, there were several paroxysms of maniacal excitement every day; and in another, that of a young lady aged twenty-three, so furious were the exacerbations that, for fear she would injure herself or others, she had to be restrained by two strong nurses, who held her while the fits lasted. The predominant type, however, was melancholia.

Sleep was deranged in every case, generally in the form of insomnia, though in three cases the tendency to somnolence was excessive. In every case the dreams were of an unpleasant character; in two there was nightmare, and in one somnambulism.

Neuralgic pains were present in seventeen of the twenty-five cases. If the upper part of the cervical region was the seat of the irritation, these pains were experienced in the scalp and face; if the lower, they were seated in the neck, the shoulders, upper part of the chest, and the upper extremities. Sometimes the pain was of a dull, burning character, and was then generally seated in the muscles of the nucha. Muscular effort always increased the suffering. In accordance with Teale's experience, it several times occurred that the neuralgia was intermittent, the paroxysms coming on about sundown and lasting through the night. In none of these cases was there anæsthesia.

Motility was interfered with in eighteen cases. Sometimes there were *fibrillary twitchings;* in five cases there were *clonic spasms* of the muscles of the face and neck; in three, *general chorea;* in two, *contractions* of the flexors of the arm on one side, so that the elbow was rigidly bent; in two, the contractions were in the flexors of the hands, and in four, of the fingers. In one case there was *complete loss of power* over the hand; in four, *aponia;* and in one, almost constant *hiccough* while the patient was awake.

Nausea was present more or less in fifteen cases, and, in one, part of every thing taken into the stomach was almost immediately rejected. *Pain* in the stomach was not met with in any case.

b. The Dorsal Region.—I found the dorsal region of the spine tender in one hundred and sixteen cases. In thirty-seven of these it was conjoined with cervical, in nineteen with lumbar tenderness, and in fifteen it was affected with the whole spine, leaving forty-five uncomplicated cases.

The most prominent symptoms in these cases were connected with the viscera, the stomach being the organ commonly involved. Thus, *gastralgia* was present in every case, *nausea* and *vomiting* in nine cases, *pyrosis* in three, *gastric flatulence* in forty, and *acidity,* as evidenced by heartburn, in twenty-six.

Next in order came the heart. There were *palpitations* in twenty-six cases, *fits of oppression,* during which the heart beat with irregularity as regarded force and rhythm, in ten cases, and *attacks of syncope* in five.

There was *difficulty of breathing* in fifteen cases, and *cough* in fifteen.

Intercostal neuralgia existed in ten, and *infra-mammary pain* in thirty-one cases.

There were no muscular spasms, contractions, or paralysis.

In the thirty-seven cases in which the dorsal tenderness was conjoined with cervical tenderness, the symptoms characteristic of each region were more or less intermingled. In two cases there was *epilepsy*, and in three *chorea paralytica*.

c. *The Lumbar Region.*—This portion of the spine exhibited tenderness in forty-nine cases. In nineteen of these it was accompanied by dorsal tenderness, in fifteen the whole spine was affected, and in fifteen the tenderness was confined to the lumbar region alone. Of these latter all were characterized by *neuralgic pains* in the lower extremities, and in three of them there were similar pains in the muscles of the back and abdomen. In six there was *spasm of the neck of the bladder*, accompanied with severe pain, and causing great difficulty of urinating, in one there was *incontinence of urine*, in five *pain in the uterus and ovaries*, and in one *neuralgia of the rectum.*

Motility was affected in eight cases. In four of these there were strong *tonic contractions* of the muscles of the lower extremities, and in four *paralysis*. In all of these there were occasional *clonic spasms* simulating chorea. Of the nineteen cases in which there was also dorsal tenderness, the symptoms were in general those characteristic of spinal irritation of both regions.

d. The whole spine was tender in fifteen cases, and so extensive was the hyperæsthesia that it was scarcely possible to press upon the most limited spot without producing pain. Of these cases the most prominent symptom in three was *epilepsy*, in one *paralysis*, sometimes of the upper and sometimes of the lower extremities, and in three *contractions* of the limbs. *Neuralgic pains*, either in the scalp, face, neck, chest, upper extremities, abdomen, and lower extremities, were present in every case, according to the part most severely affected for the time being. The heart was disordered in five cases, the stomach in ten, in three there was *difficulty of swallowing*, from alternating paralysis, and spasm of the muscles of the larynx, and in two *aphonia*.

Causes.—The most powerful predisposing cause is *sex*. Of the one hundred and fifty-six cases, one hundred and forty were females. Age is likewise influential in determining to the disorder. Of one hundred and thirty-seven cases in which I have recorded the age, seventy-two were between fifteen and twenty-five, thirty-two between twenty-five and thirty-five, fifteen under fifteen, and eighteen over thirty-five. The period of life between fifteen and twenty-five is therefore that at which spinal irritation is most apt to occur.

Hereditary influence was ascertained to exist in thirty cases.

The exciting cause of spinal irritation is not always easy to ascertain. In thirty out of one hundred and thirty-seven cases I could not, by the most careful inquiry, find any circumstance likely to have given it origin. In twenty-one it was manifestly produced by blows, falls, or strains, in twelve it was obviously caused by sexual excesses, and four by onanism. In ten there was reason to ascribe it to anxiety and grief, in two to excessive mental exertion, in twenty-one to insufficient physical exercise, in fourteen to innutritious and insufficient food, in three to over-indulgence in alcoholic liquors, and in one to the use of opium. In the remaining nineteen cases it followed exhausting diseases, such as typhoid, scarlet, and intermittent fever, dysentery, and diphtheria, and was probably directly the result of their influence.

It may also be caused by obliteration of the aorta or spinal vessels, by tumors, thrombosis, or embolism, by hæmorrhage from vessels in relation with those of the cord, or by exposure to severe cold.

In general terms, it may be said that any cause capable of reducing the powers of the system may produce spinal irritation.

Morbid Anatomy and Pathology.—I have already stated it as my opinion that the essential condition of spinal irritation is anæmia of the posterior columns of the cord. Other writers have ascribed it to inflammation, congestion, hysteria, and numerous other factors. The reasons which have induced me to arrive at this conclusion are briefly as follows: Owing to the fact that spinal irritation is not *per se* a fatal disease, we rarely have the opportunity to verify any views we may hold in regard to its pathology. In the few cases in which post-mortem examinations were made, nothing abnormal was found, a circumstance, however, far more compatible with the idea I have expressed than with any other:

1. It is a well-recognized fact that irritation is often a result of a deficient supply or a poor quality of blood. Thus headaches are frequently caused by cerebral anæmia, and are promptly relieved by increasing the amount of blood in the cerebral blood-vessels. Irritability of the mind is also a constant accompaniment. A feebly-nourished stomach rejects food, and is the seat of pain. An anæmic heart beats with great rapidity, weak muscles are affected with tremor, and an exhausted generative system is brought into a state of unnatural erethism by the slightest kind of excitation. Analogy, therefore, supports the theory I have suggested.

2. The diagnosis of diseases of the spinal cord has become so perfect that we are able to distinguish congestion, meningitis, myelitis, softening, tumors, etc., by their symptoms and by the means of research at our command. We see, therefore, that the morbid phenomena which result from such conditions are not such as we now class under the head of spinal irritation. This division of the subject will be more fully considered under the head of diagnosis.

3. I have repeatedly ascertained, by actual experience, that those agents which are known to diminish the amount of blood in the spinal vessels invariably increase the severity of the symptoms due to spinal

irritation, while they are as effectually lessened in intensity by remedies which tend to produce spinal hyperæmia.

4. The general condition of patients the subjects of spinal irritation is always below par, and the exciting causes are all such as tend to the production of asthenia.

5. The character of the symptoms points decidedly to the greater, and at times sole implication of the posterior columns. There are cases of the disorder in which there is no derangement of motility in any part of the body, and in all cases aberrations of sensibility are the prominent features. Moreover, the viscera are generally affected in their functions, a circumstance of itself strongly indicative of the situation of the lesion in the posterior columns.

These circumstances, I think, go very far toward confirming the view I have expressed, that in spinal irritation the vessels of the cord, especially those of the posterior columns, contain less blood, and that this fluid is inferior in quality to that of the organ when it is in a healthy condition. Now that the function of the sympathetic nerve, as regards its action in regulating the calibre of the blood-vessels, is so satisfactorily proven, we can partially understand how local congestions and anæmias may be superinduced. It is probable, therefore, that the original disturbance in many cases of spinal irritation resides in the sympathetic system, and the intimate anatomical relations existing between the two nervous centres are strongly in favor of this suggestion.

On the other hand, many of the phenomena of spinal irritation point strongly to the secondary involvement of the sympathetic system. It is thus that the visceral disturbances which form such prominent features are mainly to be explained.

The pathology of several others of the more striking symptoms of spinal irritation has been a subject of frequent discussion, but at the present day presents no difficulties. Thus the excitation of pain in the tissues to which the cutaneous nerves are distributed results from the law that irritation at a nervous centre induces pain at the points in which the nerves arising from that centre end. Each compound spinal nerve sends a twig to the skin contiguous to it, and these twigs terminate immediately over the spinous processes. Now, whenever an irritation is thus transmitted to the periphery, it may be reflected back to the centre whence it came, by local irritations. Thus a patient is suffering from chronic inflammation of the spinal cord, and in consequence has pain and muscular spasms in his lower extremities. An irritation applied directly to the cord increases the pain and spasms; an irritation applied to the lower extremities augments the pain in the cord, and may induce pain and spasms in distant parts of the body. Hence it is that pressure on the skin over the spinous processes not only causes cutaneous pain, but also gives rise to spinal pain, and neuralgic sensations in those nerves which come from the irritated part of the cord.

The pain existing in the cord is aggravated by percussion or muscular action. The spinal cord, it is true, is inclosed in a strong and thick, bony canal, which, however, is entirely filled by its contents. blow, therefore, on the exterior of the column causes a vibration, which is propagated through the bony structure to the cord and its membranes. If this blow be very violent, the concussion may be such as to inflict irreparable damage on the cord. When any portion of the cord is in a state of irritation, a very light blow upon the spinous processes over the disordered part, will cause severe pain, or notably add to that already present. The vertebral column is flexible, and therefore muscular action may, by producing deviations from the ordinary line followed, occasion pressure, and, in the abnormal condition of the cord, excite pain.

Diagnosis.—Recollecting that no case is to be regarded as one of spinal irritation which is not characterized by spinal tenderness, we have our diagnostic inquiries limited to the distinguishing of spinal irritation from other spinal affections. It is certainly true that the distinction has often been overlooked, and that at times there is a real difficulty in forming a correct judgment. Nevertheless, by carefully estimating all the circumstances, permanent errors of diagnosis are not likely to occur.

There are three diseases of the spinal cord which may in their earlier stages be confounded with simple spinal irritation. These are chronic myelitis, meningitis, and congestion. As the treatment of these affections is in many respects the exact reverse of that proper for spinal irritation, and as they are of far more serious character, it is important to make as early and as correct a discrimination as possible.

In both spinal irritation and myelitis there is tenderness over some part of the vertebral column, which tenderness is increased by pressure, but this tenderness is never due to hyperæsthesia of the skin, whereas in spinal irritation it often is.

In spinal irritation there is never, so far as my experience goes, anæsthesia, whereas this is a constant accompaniment of myelitis.

The contractions which take place in some cases of spinal irritation are painless, while those due to myelitis are attended with great suffering.

In myelitis there is a sensation as if a tight cord were tied around the body at the upper limit of the paralysis, a sensation which is absent in spinal irritation. It is true that Mr. Teale has described several cases which he classed as spinal irritation and in which the sensation of constriction was present, but careful examination of the histories leaves scarcely a doubt that these were really cases of myelitis.

The bladder is never paralyzed in spinal irritation, whereas in myelitis it generally is, if the inflammation be located in the lower dorsal region of the cord. The same is true of the sphincter ani. Myelitis is always productive of paralysis, and there is always more or less atrophy of the paralyzed muscles. Spinal irritation seldom gives rise to paralysis, which, when it does result, is always incomplete, and is never productive of atrophy.

The progress of myelitis is generally, unless arrested by appropriate treatment, toward a worse condition, whereas no such tendency is manifested by spinal irritation.

From spinal meningitis, spinal irritation is distinguished by the circumstances that in the former disease there are constant painful spasms of the muscles of the back, pain in the cord, and no spinal tenderness increased by pressure.

From congestion of the spinal cord and its membranes, spinal irritation is sufficiently distinguished by the facts that there is generally little or no pain in the cord in the first-named affection, and no spinal tenderness. In congestion, likewise, the paralysis and other symptoms are always worse after the patient has been lying down, while in spinal irritation the recumbent position always alleviates the condition.

Another means, which in doubtful cases will invariably lead to a correct diagnosis, is afforded by the known effects of certain medicines. Thus spinal irritation is, as I have several times ascertained, made worse by the administration of ergot, while each one of the other diseases I have named is alleviated. The reverse is true of strychnia, which in all cases aggravates the symptoms of myelitis, meningitis, or congestion, while it is an efficient means of cure in spinal irritation. An hypodermic injection of the thirtieth of a grain is sufficient to settle the matter in cases where the diagnosis is of difficult formation.

The flatulence, eructations, and vomiting, are very symptomatic of spinal irritation, while they are rarely phenomena of either of the other affections.

One other disease is liable to be confounded with spinal irritation, and that is angular curvature, in which there is spinal tenderness increased by pressure. The facts, however, that strumous disease of the vertebræ generally occurs in children, that the scrofulous diathesis is always present, that an angular prominence can be detected by careful examination, that the paralysis progressively becomes more profound, that the constitutional effects are more severe, are sufficient, even in doubtful cases, to guide to a correct diagnosis.

Prognosis.—The prognosis in cases of spinal irritation is generally favorable. In fact, so far as my experience extends, I have never seen a case which entirely resisted treatment, and very few in which a cure was not ultimately effected. When remedies suitable for the difficulty do not prove successful, it is because the patient does not steadfastly persevere in their use.

Of the one hundred and fifty-six cases forming the basis of this chapter, one hundred and thirty-three were thoroughly cured, ten were lost sight of soon after treatment was commenced, but were materially improved, and thirteen were relieved for the time being, but continued to have relapses.

Treatment.—The principles of treatment applicable to spinal irritation are four:

1. To remove the cause.
2. To improve the general tone of the system.
3. To increase the amount of blood in the spinal cord, and improve the nutrition of this organ.
4. To set up a counter-irritant action in the vicinity of the disordered region of the cord.

In regard to the first indication, I have nothing special to say. The cause once ascertained, common-sense would dictate its removal as speedily and as effectually as possible, by the proper means according to its character.

The second indication is to be met by tonics, such as quinine and iron, and especially stimulants judiciously administered. I am as well convinced of the general applicability of alcohol in some form, in the treatment of spinal irritation, as I am of any thing. Whiskey, brandy, and rum, are to be preferred on account of their less liability to disagree with the stomach, and as containing a greater percentage of alcohol than vinous or malt liquors. Among the tonics the preparations of zinc are valuable, and I think the oxide is to be preferred. Cod-liver oil is also of great service.

The third indication is easily fulfilled by strychnia, phosphorus, phosphoric acid, and opium. The two first-named remedies may be very satisfactorily combined in a pill containing half a grain of extract of nux-vomica and the tenth of a grain of the phosphide of zinc, which may be given three times a day. Strychnia may also be given by solution of the sulphate in dilute phosphoric acid, and in doses of about the thirty-second of a grain to half a drachm of the acid. The beneficial effects of these remedies are perceived in a few days. Opium is especially useful in those cases in which there are contractions of the limbs, and here its action is, of course, not solely that of an agent increasing the amount of blood in the cord. I prefer to give it either in the form of suppositories, composed each of half a grain of the aqueous extract and a sufficient quantity of the butter of cacao, or by hypodermic injection of morphia. I have frequently seen contractions, which had persisted with obstinacy for several weeks, relax in a few minutes under the influence of opium thus administered.

The application of hot water to the spine is also an admirable adjuvant. It should be used as hot as can be borne. Nothing is better for the purpose than Dr. Chapman's India-rubber bags.

But there is a remedy which apparently either contracts or enlarges the diameter of the blood-vessels, and which is more efficacious in removing spinal irritation than any other with which I am acquainted, and that is the direct galvanic current. The method I follow in cases of spinal irritation is the application of either pole at some point above the seat of the pain, and the other at a point an equal distance below. The current thus brought to bear upon the cord, seemingly conduces to the dilatation of the blood-vessels and the improvement of the nutrition of the cord. The current should not be passed at any one *séance* for more than fifteen minutes, and no one application should last longer than three or four minutes. For the relief of the spinal tenderness one pole should be applied directly to the painful part, and the other to a point distant laterally from it a few inches.

The fourth indication is one of great importance, and, when properly carried into effect, a cure will often result in slight cases without any other means of treatment being employed. The *rationale* of the action of counter-irritants in this and similar derangements is by no means clearly understood. It is a question which I do not, however, propose to discuss. Of counter-irritants my experience leads me decidedly to the employment of blisters in preference to any others. They should be applied to the skin, immediately over the painful part of the spine, and should be renewed as often as may be necessary. Tartar-emetic ointment, though useful, is more painful and I think not so efficacious as blisters. Dry cups are more admissible, and almost always do good. They should be applied on each side of the spinous processes for an extent of four or five inches above and below the painful spot. Leeches, or any other means for the abstraction of blood, are, according to my experience, always prejudicial.

Besides these therapeutical means, there are others of a more strictly hygienic character, which cannot be overlooked. Thus the food should be of a highly-nutritious character, moderate physical exercise should be taken, and as much time as possible should be spent in the open air.

Patients almost always feel more comfortable in the recumbent position than any other, because thereby the blood is allowed to settle in the spinal vessels. They should not therefore be prevented lying down during the greater part of the day, but at the same time they should be encouraged to take exercise, and especially so when there is any loss of power in the lower extremities. The induced or faradaic current is almost always of service, when applied to the affected muscles, and the direct is of great efficacy when passed through neuralgic nervous trunks.

In illustration of the views inculcated in this chapter, I append the following details of cases:

CASE I. *Irritation of the Cervical Region of the Spinal Cord.*—Mrs. J. S. consulted me, May 7, 1868, for what she had been informed was a cerebral disorder. The patient was thirty-eight years of age, had had five children, and had always enjoyed good health till two years previously, when she had been thrown from her carriage. She was not stunned or otherwise seriously injured. Soon after the accident she noticed a rumbling noise in one ear, and in a few days subsequently the other ear became similarly affected. About the same time there were flashes of light before the eyes, and a dull, heavy pain in this point of the head. Vertigo was also frequently present. There was insomnia, and when she did sleep she was very apt to be attacked with nightmare.

These symptoms continued to annoy her for several months, without, however, compelling her to seek for medical advice, until at last she had a seizure which was certainly epileptic in its character. This was followed with disturbance of vision, and intense neuralgia of the fifth pair of nerves. She now placed herself under the charge of a physician in a neighboring city, where she was then residing, who diagnosticated a tumor of the brain, and gave an unfavorable opinion as to the ultimate result. He, however, advised the use of iodide of potassium. She took this in large doses faithfully for three months—during which period she had two more epileptic attacks—without perceiving any benefit, and then she went to Europe. While there she consulted a number of physicians and surgeons of eminence, all of whom gave a very guarded prognosis. By the advice of several of these she took the bromide of potassium, with, at first, some advantage, but this was eventually lost, and her symptoms became as severe as before. She had several epileptic paroxysms during the four months she was taking the bromide. Finally, she traveled through Germany and Italy, and, still obtaining no relief, returned home. I saw her a few days after her arrival. She was then suffering from facial neuralgia, excessive tenderness of the scalp, so that she could not have her hair brushed without enduring great pain, obscureness of vision, pain in the eyeballs, redness of the conjunctivæ, vertigo almost constantly, great mental irritability, amounting at times to positive insanity; wakefulness, nightmare, and contraction of the fingers, the nails being strongly pressed against the palm of the hand.

Ophthalmoscopic examination showed dilatation of the retinal vessels, arterial and venous pulsation, and congestion of the optic disks of both eyes. The pupils of both eyes were contracted.

Perhaps I should not have suspected any spinal disorder, if she had not herself called my attention to a pain which she said she constantly felt between the shoulders. I therefore examined the upper part of the spine very carefully, and found deep-seated pain developed by percussion over the seventh cervical vertebra, and great hyperæsthesia of the skin in the same region. Her symptoms were not those in the least indicative of congestion of the cord or its membranes, of meningitis, or myelitis, and the apparent severity of the cerebral symptoms, and the general good condition of her mind and sensorial and motor functions, were so incompatible, that I could not, upon reflection, bring myself to the belief that she was affected with any organic disease of the brain. My inquiries and examinations all led me to the conclusion that she was laboring under spinal irritation of the lower cervical region.

I therefore prescribed for her five drops of the phosphorated oil three times a day, applied a blister to the painful spot, and daily passed the direct galvanic current through the cord, by applying the negative pole to the fifth cervical, and the positive to the sixth dorsal. My object was, not only to improve the nutrition of the cord, but also, by irritation of the sympathetic, to contract the vessels of the brain. Budge and Waller had shown, several years previously, that, when that portion of the spinal cord situated between the seventh cervical and sixth dorsal vertebræ is acted upon by the galvanic current, the pupils are dilated. Now, dilatation of the pupils is produced by excitation of the sympathetic, and excitation of the sympathetic, within the limits mentioned, likewise causes contraction of the vessels of the brain, as can readily be seen by ophthalmoscopic examination while the current is passing.

Under the influence of this treatment the amendment was rapid, and at the end of three months she was entirely cured. It was necessary, however, to apply eleven blisters.

CASE II. *Irritation of the Cervical Region of the Cord.*—M. S., a gentleman of sedentary habits, consulted me, August, 1867, for intense headache and facial neuralgia, with which he had suffered for several months. The disease had come on gradually, and, although now never entirely absent, was paroxysmal in its character, being more severe at night than through the day. The external pain followed the course of the fifth pair of nerves through all its branches; the internal was fixed in the posterior part of the head, and was evidently due to cerebral anæmia, as it was relieved by stimulants and by holding the head in a dependent position. Vertigo was frequently present, and the disposition to sleep was excessive, though, owing to the pain, it could not be indulged in for more than a few minutes at a time. Nausea was occasionally a symptom, but never to the extent of being followed by vomiting.

On examining the spine of this gentleman, I found tenderness over the fourth and seventh cervical vertebræ. Two blisters were at once applied, and Aitken's syrup of the phosphate of iron, quinine, and strychnia, administered. From the first, improvement was manifested, and in less than a month the cure was complete.

CASE III. *Irritation of the Dorsal Region of the Spinal Cord.*—Mrs. J. B., aged twenty-four, consulted me, March, 1868, for obstinate vomiting, and neuralgic pains in the left breast. She was thin, pale, and anæmic, and had suffered for over a year. She also complained of a dull, aching pain in the middle of the back, which was increased by even moderate physical exercise. The vomiting took place regularly after every meal, and even water was at once thrown up. She was under the impression that the disorder was the result of exposure t several hours to very severe cold while in an open boat.

Recognizing, at once, the fact that the main difficulty lay in t cord, I carefully examined the whole spine, and found excessive tend ness over the spinous processes of the sixth, seventh, and eighth dor vertebræ. There was also deep-seated spinal pain developed by perc sion.

I ordered the application of a blister, and the internal use of s quantities, frequently repeated, of milk-punch (one ounce of brand three of milk). The first wineglassful was at once rejected, and s was a tablespoonful which she took half an hour subsequently. I then reduced the quantity to a teaspoonful every half-hour. This was retained, and was the first nutriment of any kind which, for nearly eleven months, had not been rejected wholly or in part.

The next day I found that the blister had drawn well, and that the nausea and vomiting were greatly diminished, as were likewise the neuralgic pains. A teaspoonful of the following mixture was then directed to be taken three times a day, immediately after meals: ℞. Strychniæ sulph., gr. j; ferri pyrophosph., quiniæ sulph., āā ʒ ss; acid. phosph. dil., syrupi zingiberis, āā ℥ ij. M. ft. mist. The milk-punch was still continued, but, in treble the dose, less frequently given.

Gradually all the symptoms decreased in violence, and at the end of two weeks she was enabled to retain a moderate quantity of food at each meal. Any excess was still, however, followed by vomiting. She had increased five pounds in weight, and was greatly improved in personal appearance.

In two months she had gained twenty-one pounds, and was as well as she had ever been in her life. The spinal tenderness had entirely disappeared; seven blisters were applied in all.

Case IV. *Irritation of the Dorsal Region of the Spinal Cord.*—Mrs. W. had for more than three years suffered from spasmodic movements of the upper extremities, not distinguishable from those of true chorea, which occasionally were followed by contractions of the flexors of the wrists and fingers. There were also infra-mammary pain, eructations, and vomiting. When she came under my care, June 22, 1869, she was reduced to almost a skeleton, and was suffering, in addition to the symptoms above mentioned, from acute pain in the back. This pain she informed me had not been ordinarily very severe, but was, nevertheless, constantly present. On examination I found tenderness over the first, second, and third dorsal vertebræ. I at once applied the constant galvanic current in a manner already described, and continued it for five minutes, with the effect of mitigating the pain in the spine and the nausea. The ensuing day I repeated the application, and in addition prescribed the mixture given in Case III. She retained it on her stomach, as she did the food which she ate that day. Brandy in ounce-doses was given with her lunch and dinner. The galvanism was continued daily for eighteen days, at the end of which time she was free from pain, from the spasms, and from the vomiting. Her appearance was immensely improved, and she had increased seven pounds in weight. The galvanism was now discontinued, but the strychnia mixture and the brandy were persevered with for over a month longer. She was then well.

CASE V. *Irritation of the Lumbar Region of the Spinal Cord.*—E. T., an unmarried lady, aged twenty-nine, consulted me, August, 1869, for paralysis of the lower extremities, attended with spinal tenderness and abdominal pains. She had been treated for inflammation of the spinal cord, had been cupped, leeched, and had had an issue made over the seat of the pain.

When I first saw her she was unable to walk, having been in this condition for several months. As she sat in her chair, she could readily move her legs in any desired direction, but to bear her weight upon them was an utter impossibility. There was no alteration of sensibility. Her general appearance was not anæmic, nor was she in the least degree hysterical. Upon careful examination, I was unable to find any reason to induce the belief that she was laboring under spinal congestion, meningitis, or myelitis, or that there was softening of, or pressure upon, the cord. I, however, discovered great tenderness over the first and second lumbar vertebræ, and found that strong pressure in this region induced deep-seated spinal pain and sharp neuralgic sensations along the course of the crural nerves.

Regarding the case as one of pure spinal irritation, I applied the constant galvanic current to the back every alternate day, and administered the following prescription : ℞. Zinci phosphidi, gr. iij ; ext. nucis vom., gr. xv. M. ft. in pil. no. xxx. Dose, one three times a day. I likewise directed the application, to the painful part of the spine, of flannel, wrung out of spirits of turpentine, to be continued daily till redness and decided smarting were produced. A full and nutritious diet, with ale, was enjoined. Under this treatment she improved so rapidly in every respect that in twenty-three days she was able to walk with a cane, and in a few days more than a month was well, being in as good health, according to her own report, as she had ever enjoyed in her life.

ANÆMIA OF THE ANTERO-LATERAL COLUMNS OF THE CORD.

The phenomena which in my opinion are the result of an anæmic condition of the antero-lateral columns of the spinal cord have hitherto been classed under the heads of spinal paresis, functional paralysis, reflex paralysis, inhibitory paralysis, paralysis from peripheral irritation, etc. Several of these names are applied with reference to the causes, others with reference to the symptoms, but none to the lesion.

Symptoms.—The most prominent symptom of anæmia of the ant ro-lateral columns of the spinal cord is paralysis of motion in tho parts of the body which derive their nerves from the affected porti of the cord, and in many cases of those below the seat of the lesi This paralysis is incomplete, the patient, if the lower extremities a affected, being able to walk, though he does so with difficulty. It noticed, too, that some muscles are more apt to be paralyzed t others, the tibialis anticus and the peroneal group rarely escaping.

In the great majority of cases the paralysis is confined to the lo extremities, constituting paraplegia. The reason for this is, that the anæmic condition of the cord which causes the paralysis is more frequently excited by irritation transmitted from the genito-urinary and digestive organs than from any others.

Spasmodic contractions of the paralyzed muscles are not often met with, though occasionally there are slight twitchings, fibrillary in their character.

It is rarely the case that the paralysis extends, as it does in that which results from congestion of the cord. The affection usually supervenes suddenly, and is about as severe in the beginning as at any subsequent period.

The bladder and rectum are very rarely involved as a consequence of the spinal lesion, though disease of either of these organs often causes anæmia of the antero-lateral columns of the cord. In a few cases, however, I have witnessed both paralysis of the bladder and of the sphincter coming on late in the course of the disease, and evidently dependent on it.

Electro-muscular irritability is rarely impaired. Reflex excitability is also generally unaffected. In the worst cases, tickling the sole of the foot will cause the leg to be drawn up, even against the volition of the patient.

Disorders of sensibility are not prominent features in anæmia of the antero-lateral columns of the spinal cord. Locally there is very rarely pain, and in the paralyzed parts there is neither anæsthesia, hyperæsthesia, nor abnormal sensations of any kind. There is never, in the uncomplicated affection, the sensation of constriction about any part of the body. The stomach and bowels are not often affected, unless there is at the same time some degree of anæmia of the posterior columns. But in one very interesting case, occurring in a lady of this city, and produced by exposure to extreme cold while crossing to Governor's Island in an open boat, there were vomiting every time food was taken into the stomach, and the most obstinate constipation I have ever witnessed. It very frequently happened that this lady had no operation from her bowels for over a month.

Causes.—Anæmia of the antero-lateral columns of the spinal cord may be produced by any cause capable of interrupting the flow of blood to the region in question, of lessening the calibre of its autocthonous arteries, or of so lowering the quality of the blood as to unfit it for the purposes of nutrition.

Thus it may be caused—though not without the implication of the posterior columns—by abdominal tumors compressing the aorta, or by disease of this vessel, leading to partial or complete obliteration; by thrombosis or embolism of the spinal arteries; or by direct loss of blood from vessels supplying the cord, or deriving their blood from the spinal vessels.

The calibre of the intra-spinal vessels may be lessened through the influence of extreme cold, and anæmia of the antero-lateral columns thus be induced. Several cases of this kind have come under my care, in which paraplegia has supervened suddenly during or after exposure to very low temperature, especially when combined with a moist state of the atmosphere. Lying on damp ground has caused it in a number of instances.

It not unfrequently follows exhausting diseases of various kinds. I have known it to supervene on dysentery, diarrhœa, cholera, typhoid fever, typhus, diphtheria, and several other affections.

But the most common cause of the disorder is undoubtedly peripheral irritation, and this is very frequently an affection of the genito-urinary organs. My friend Dr. S. Weir Mitchell [1] has written very exhaustively on this subject, and has shown the relation which exists between the different paralyses now usually called reflex, and injuries of nerves. Under the head of pathology I shall have occasion to return to Dr. Mitchell's valuable contributions.

Diagnosis.—Anæmia of the antero-lateral columns of the cord is distinguished from congestion by the facts that the symptoms are mitigated by the recumbent position instead of being increased in violence, as in the latter affection; that the paralysis shows no tendency to become more severe, and that, when the bladder or rectum is involved, the derangement of either viscus precedes the paralysis.

From anæmia of the posterior columns, it is diagnosticated by the fact that the more obvious symptoms are related to motility, sensibility not being involved, while in the former the reverse is the case.

The diagnosis from myelitis will be pointed out when inflammation of the cord is under consideration.

Prognosis.—The probability of a favorable termination is great. In fact, no affection of the cord is so susceptible of cure when there is no mechanical obstruction in the aorta or spinal arteries. But this opinion is expressed with the understanding that the cause must first be removed. So long as this continues in action, anæmia of the antero-lateral columns of the cord is a very obstinate affection. When the arteries are obstructed, then, as in the brain under like conditions, softening of the cord may take place.

---

[1] Circular No. 6, 1864, Surgeon-General's Office. "Reflex Paralysis," by Drs. Mitchell, Morehouse, and Keen. Also "Wounds and Injuries of Nerves by the same," Philadelphia, 1864. Also "Paralysis from Peripheral Irritation," by Dr. Mitchell, *New York Medical Journal*, February, 1866.

Morbid Anatomy and Pathology.—Post-mortem examination, of persons who have suffered with symptoms indicative of what I consider to be anæmia of the antero-lateral columns of the cord, does not reveal the existence of any material spinal lesion. The reason for this is, that anæmia of the cord is, in the nature of things, a very difficult disease to detect, and cannot be definitely made out, unless the capillaries are measured under the microscope.

But it is this very absence of obvious lesions which indicates very positively the existence of anæmia, and the character of the symptoms shows that the antero-lateral columns are its seat.

Several varieties of paralysis result from anæmia of the antero-lateral columns. Classing these as Mitchell [1] has done, from their apparent causes, we find that there are—

1. Paralyses arising during disease of the genito-urinary organs.
2. Those which occur during or just after dysenteries, diarrhœas, super-purgation, or in connection with worms.
3. Such as arise during or after pneumonia or pleurisy.
4. Such as are seemingly brought on by dentition.
5. The paralysis of diphtheria, fevers, and eruptive disorders.
6. Such as seems to be occasioned by cold, or by cold and moisture.
7. Paralysis due to external injury.

To this list may be added—

8. Paralysis resulting from certain medicines and drugs.
9. Paralysis due to great emotional disturbance.

Many cases of each of these varieties of paralysis have come under my notice, and there are few medical practitioners who have not witnessed instances referable to one or more of the foregoing categories. The principal theories of their immediate cause are—

1. That of Mr. Stanley,[2] by which certain varieties of paralysis are attributed to the transmissal of an irritation from a diseased organ to the spinal cord, whence it is reflected to the muscles as paralysis.

This is no explanation at all, and leaves the condition of the cord out of consideration. There is no proof whatever that an irritation can, without causing change in the structure of the nervous centre, induce either paralysis of motion or of sensation.

2. That of Dr. Brown-Séquard,[3] which ascribes the affections in

---

[1] "Paralysis from Peripheral Irritation, with Reports of Cases," *New York Medical Journal*, February, 1866, p. 323.

[2] "On Irritation of the Spinal Cord and its Nerves in Connection with Disease of the Kidneys," "Medico-Chirurgical Transactions," vol. xviii., p. 260.

[3] Lectures on the "Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities," Philadelphia, 1861.

question to a lesion of the cord, consisting in a spasm of the spinal vessels by which their calibre is diminished. This spasm is, according to this eminent neurologist, the result of a peripheral irritation transmitted through the nerves coming from a diseased organ or part of the body, to the vaso-motor nerves of the portion of the cord giving origin to these nerves.

This was, so far as I have been able to ascertain, the first attempt to designate the character of the lesion, which, as will be at once perceived, is anæmia. That anæmia can be induced by peripheral irritation is, I think, well established. But though this theory accounts for many cases of spinal paralysis, such as are now under notice, it will not embrace all, for we may have anæmia and consequent loss of motor power resulting from other causes than irritation. Moreover, Dr. Brown-Séquard did not fix the lesion in the antero-lateral columns, nor associate the symptoms with any derangement in the structure of this region of the cord.

3. Dr. Mitchell, in the paper to which I have already referred, divides the several kinds of paralysis mentioned into three classes: those which are asserted to be due to disease of the genito-urinary system, a cause which he denies *in toto*; those which are said to be produced by peripheral irritation of the intestinal canal, an influence which he also in great part denies; and those which follow wounds and injuries of nerves.

Dr. Mitchell rejects altogether the reflex theory of Dr. Brown-Séquard, and says:

"If I were now to sum up the probabilities in the way of causation of palsies peripherally induced, I should be disposed to refer some cases to exhaustion from too constant or excessive exercise of normal functions, and others to irritation from disease or injury, and to consequent exhaustion of the centres; while, as regards the intervention of vascular agency, I should reject the idea of prolonged vasal spasm, and consider it possible that in some instances over-excitation might result in dilatation of the vessels, in which case some material lesion would surely result if the condition in question were of long continuance."

While not prepared to accept Dr. Mitchell's views in their entirety, they are, in my opinion, perfectly in accordance with the doctrine of anæmia of the antero-lateral columns. As to whether this anæmia is the result of spasm of the spinal vessels, or exhaustion, is a question which, for the present at least, is not definitely settled. My own opinion is that paralyses of apparently peripheral origin are referable to anæmia, produced in some cases by vaso-motor spasm, and in others by nervous exhaustion.

The experiments of Küssmaul and Tenner[1] are perfectly conclusive as to the effects of cutting off the supply of blood to the spinal cord.

---

[1] "The Nature and Origin of Epileptiform Convulsions caused by Profuse Bleeding, etc." New Sydenham Society Translations, London, 1859, p. 53, *et seq.*

These observers compressed the aorta in rabbits so completely that not a drop of blood could reach the spinal cord below the point of occlusion. The consequence was, that there was complete paralysis of all the muscles receiving their nervous influence from the anæmic portion of the cord. The possibility, therefore, of spinal anæmia producing paralysis, is beyond doubt. In these experiments, however, the blood was of course shut off from both the anterior and posterior columns, and therefore the phenomena were not those of simple motor paralysis.

M. Vulpian[1] has recently discussed, very thoroughly, the several questions connected with the pathology of reflex paralysis. By an experiment, which consisted in faradizing a communicating branch of the intra-thoracic chain of ganglia, a decided contraction was seen to take place in the vessels of the spinal cord at the point of origin of the intercostal nerve in relation with the irritated branch. When the faradization was intermitted, the vessels returned to their former size—or, perhaps, even became a little larger than was natural. This experiment was therefore followed by a result similar to that recorded by Brown-Séquard, who says:[2] "A contraction of blood-vessels in the spinal cord *I have seen* (in the vessels of the pia mater) take place under my eyes, when a tightened ligature was applied on the hilus of the kidney, irritating the renal nerves, or when a similar operation was performed on the blood-vessels and nerves of the suprarenal capsules. Generally in these cases the contraction is much more evident on the side of the cord corresponding with the side of the irritated nerves, which fact is in harmony with another and not rare one, observed first by Combaire (as regards the kidney), and often seen by me after the extirpation of one kidney, or one suprarenal capsule—i. e., paralysis of the corresponding lower limb."

M. Vulpian admits that anæmia of the spinal cord causes with great rapidity the abolition of the medullary functions. The fact is established by experiments consisting in the obliteration of the spinal vessels by substances injected into them. Thus Flourens,[3] many years ago, injected the powder of lycopodium into the crural artery of a dog, taking care to throw the substance with some force into the artery against the current of the circulation, so that it entered the abdominal aorta and was distributed to the spinal vessels. The powder occluded the more minute of the arteries, and a localized anæmia of the spinal cord was thus produced. The result was, that the posterior extremities of the animal were almost immediately paralyzed.

Feltz[4] injected finely-powdered charcoal into the right crural artery of a dog, so that the injection passed into the inferior part of the abdominal aorta. The animal was at once paralyzed in the right posterior extremity, and shortly afterward, in the corresponding limb of the opposite side. After death, particles of the powder were found in the spinal arteries.

---

[1] "Leçons sur l'appareil vaso-moteur," etc., Paris, 1875, tome ii., p. 48, *et seq.*

[2] "Lectures on the Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities," Philadelphia, 1861, p. 24.

[3] "Comptes rendus de l'Académie des Sciences," 1847, p. 905.

[4] "Traité clinique et expérimental des embolies capillaires," Paris, 1870, p. 186.

Vulpian[1] has several times repeated these experiments, using the powder of lycopodium, and has invariably found the animals become almost instantly paraplegic.

Nevertheless, he is not sure that the paralyses, called reflex, are the result of spinal anæmia; on the contrary, he doubts if, in reality, there are any such affections. He is disposed to think that they are to be classed in several categories; one embracing cases in which there is a definite lesion of the cord; another, those cases which occur in hysterical, hypochondriacal, and epileptic persons, from irritations existing in distant parts of the body, and which he calls paralyses of peripheral origin; and a third, comprehending all instances which cannot be included in either of these classes, and especially embracing the cases due to the action of cold on the surface of the body.

M. Vulpian's chief objection to the theory of spinal anæmia is that, when the arteries are occluded by artificial emboli, softening of the cord takes place. This is doubtless true in the majority of cases, but certainly not in all, for in the dog, which was the subject of Feltz's experiment, life was prolonged for two days, and it is expressly stated that the cord was not softened. Moreover, in the anæmia produced by peripheral irritations the vessels are not entirely closed. Their calibre is simply diminished; some blood reaches the cord, but this is not sufficient for the full performance of its functions. If softening were the invariable result of a lessened supply of blood to an organ, we should meet with it constantly in cases of general cerebral anæmia from any one of the many causes capable of producing that condition.

Now, I have repeatedly performed Flourens's experiment, both with powdered lycopodium and charcoal, and have never failed to obtain paralysis of the posterior extremities. It is true the loss of power was permanent, remaining in each case during the life of the animal, but such a result is, of course, to be expected, for it is impossible to get rid of the substances occluding the vessels. In the anæmia produced by reflex irritation or spinal exhaustion, the possibility of removing the cause, overcoming the vaso-motor spasm, or improving the nutrition of the cord, places the condition entirely in a different line from that due to mechanical occlusion of the vessels, except as regards the one point of anæmia, and even here the difference is great, for in the former the supply of blood is merely lessened, while in the latter it is cut off altogether.

In practice we often find that the anæmia is not restricted to either set of columns, and that the symptoms are accordingly those of motor paralysis, aberrations of sensibility, and functional disturbances in various organs, such as we have just considered as being caused by anæmia of the posterior columns.

---

[1] *Op. cit.*, p. 53.

Treatment.—The treatment is similar in general features to that applicable to anæmia of the posterior columns already considered, though there is not the same benefit to be derived from counter-irritation. The indications, therefore, are to remove the cause, to improve the general tone of the system, and to increase the amount of blood in the spinal vessels.

So far as the first indication is concerned, it very often happens that its fulfillment is sufficient for the entire removal of the anæmia, and the disappearance of the consequent paralysis. This is especially the case as regards those instances which are due to peripheral irritations of various kinds. Within the last few days a young lady, aged twelve, was brought to me by her mother to be treated for paraplegia, which had developed very suddenly. There was no evidence of serious organic difficulty, and no apparent cause of peripheral irritation. Her symptoms, however, all pointed to anæmia of the antero-lateral columns, and, on the principle of exclusion, I thought it probable there might be worms in the alimentary canal. I therefore administered several doses of santonine, followed by castor-oil. A number of lumbrici were discharged, and the paralysis disappeared in the night as suddenly as it had arisen.

In another case, a gentleman was rendered paraplegic soon after contracting a catarrhal inflammation of the bladder. The bladder affection was disregarded by his physician, and energetic means were used against the paralysis, but without effect. I suggested the expediency of suspending the administration of the strychnia and the application of counter-irritants to the spine, and directing attention to the cure of the disorder of the bladder. This was done, and, at the same rate as the inflammation yielded to the treatment, the paraplegia disappeared.

The general tone of the system is to be improved by such measures as were recommended for the accomplishment of the same end in anæmia of the posterior columns.

For fulfilling the third indication, strychnia and phosphorus are preferable to any internal remedies. I usually prescribe them together in doses of the tenth of a grain of the phosphide of zinc, with from a third to a half a grain of the extract of nux-vomica in pill, to be taken three times a day. Lately, however, I have pursued the practice of giving the strychnia in gradually-increasing doses till there is evidence of its characteristic physiological effects being produced. Two grains of the sulphate of strychnia are to be dissolved in an ounce of water, and ten minims, containing one twenty-fourth of a grain of strychnia, given three times a day; the next day eleven minims are administered for each dose, the next twelve, and so on till, as often happens, the paralysis yields, or till the reflex excitability of the legs is increased, or stiffness of their muscles or those of the nucha is induced. In either of these latter events the administration must be stopped for a day, and then the original dose of ten minims be given and increased as before. There is, according to my experience, no medication so effectual in all those forms of paralysis called reflex, inhibitory, functional, etc., and which, in my opinion, result from anæmia of the antero-lateral columns of the cord, as this with strychnia. It requires care and prudence, and, if these qualities be exercised, is perfectly safe. It very generally happens that, before the patient reaches thirty minims (one-eighth of a grain) for a dose, the paralysis begins to yield. In one case, however, due to exposure to severe cold, I was obliged to carry the dose to sixty minims—equal to one-fourth of a grain of strychnia—before the excitability of the cord was increased, or any signs of the paralysis yielding were observed. The patient recovered after taking three-quarters of a grain of strychnia daily for over two weeks.

In a very remarkable case recently under my care, sent to me by Dr. Brooks, of Cleveland, the paralysis following diphtheria affected both arms and both legs, and was evidently increasing daily in intensity. At last, the patient could scarcely move a muscle of either extremity, and was, of course, unable to walk. Strychnia was administered according to the manner above described, and also by hypodermic injections. Amendment was slow but steady, and I sent him home, where, under Dr. Brooks's care, who boldly carried out the treatment, he entirely recovered. At no time, while under my observation, was there any evidence of strychnization, although large doses of the remedy, amounting at one time to a grain a day, were given. The irritability of the cord appeared to be entirely abolished. Neither the bladder nor rectum was paralyzed, and cutaneous sensibility was scarcely impaired.

The only local application which is decidedly beneficial in anæmia of the antero-lateral columns is the constant galvanic current, which should be used in the manner recommended for anæmia of the posterior columns.

As regards the paralyzed muscles, the induced or faradaic current is useful in keeping them exercised, and thus preserving their nutrition. Friction and kneading exercise a like effect.

In those cases of spinal anæmia due to obstruction of the aorta, or occlusion of spinal vessels by emboli, no specific treatment is of any avail.

CHAPTER III.

SPINAL HÆMORRHAGE—SPINAL MENINGEAL HÆMORRHAGE.

THESE two conditions having a common cause, being often associated, and having a general resemblance to each other, may properly be considered together.

Symptoms.—A hæmorrhage into the substance of the spinal cord is characterized by pain at the seat of the lesion, and by derangements of sensibility and of the power of motion in all those parts of the body below. These consist ordinarily of anæsthesia and loss of motility, but occasionally there are hyperæsthesia and spasms. In the great majority of cases the bladder and its sphincter and the sphincter ani are also paralyzed. Obstinate priapism is an occasional symptom. Reflex excitability and electro-muscular contractility are soon impaired or altogether lost.

An elevation of temperature is said[1] to take place in the paralyzed parts, and the formation of sloughs on the sacrum and other parts exposed to pressure is a frequent occurrence. In a case under my own observation, a man fell from a scaffold upon which he was working, and struck the small of his back against a projecting beam. He was taken up paraplegic, and within six hours three large sloughs, one over the sacrum, and one over each hip, made their appearance. Sensibility was entirely abolished in the paralyzed limbs. The bladder and rectum were completely paralyzed, and death ensued on the fifth day. Post-mortem examination discovered a clot in the substance of the cord, extending from the tenth dorsal to the fifth lumbar vertebra, and involving both the white and the gray substance. There was neither fracture nor luxation.

Hæmorrhage into the substance of the cord may be either rapidly or slowly developed. In the former case it generally terminates fatally in a few days or even hours; in the latter, life may be prolonged for several months, or may be preserved, with more or less paralysis of motion or sensibility, or, as is generally the case, of both in the parts below the seat of the lesion.

If the seat of the hæmorrhage be high up in the neck, death is almost instantaneous from the paralysis of the phrenic nerve.

When the lesion is meningeal, the symptoms are not generally so rapidly developed as when it is situated in the substance of the cord. The pain is greater and there is a more decided tendency to spasmodic jerkings in the limbs receiving their nerves from the part of the cord below the extravasation. Occasionally the convulsive movements are general, and, according to Hayem, are more marked in the paralyzed than in the non-paralyzed limbs. Hyperæsthesia may alternate with anæsthesia, or this latter may alone be present.

---

[1] Hayem, "Des hémorrhagies intra-rachidiennes," Paris, 1872, p. 186.

The extent of motor paralysis is very variable, both as regards intensity and diffusion. Sometimes all the muscles below the seat of the lesion are more or less paralyzed; at others, some muscles altogether escape. I have a patient at the present time under treatment who has, in consequence of a spinal hæmorrhage, probably meningeal, lost sensation in a small region of skin over the glutei muscles, and sensation and motion in all the tissues below both knees. Sensation and motion are intact in all other parts of the lower extremities. The bladder is unaffected, but there is very obstinate constipation.

Reflex excitability is often exaggerated, and the electro-muscular contractility increased in the early stage; but, if the patient survives the immediate effects of the lesion, both these faculties become impaired. Meningeal hæmorrhage taking place above the third cervical vertebra may be speedily fatal, from the interruption to respiration due to paralysis of the phrenic nerve.

Causes.—Spinal hæmorrhage, either in the substance of the cord or of the membranes, is generally the result of injury. Thus it may be caused by blows on the vertebral column, by falls, or by gunshot, or by wounds with penetrating instruments. It may also be produced by tetanus and by the rupture of aneurisms, but is in either of these cases meningeal.

Excessive fatigue, the suppression of the menstrual flow, undue venereal indulgence, alcoholism, yellow fever, typhoid fever, disease of the vertebræ, and the toxic influence of strychnia, have also been alleged as causes. In many cases not traumatic the immediate cause is not known.

The male sex appears to be much more liable than the female. Of nineteen cases of hæmorrhage into the substance of the cord analyzed by Gintrac,[1] fifteen were in males.

Diagnosis.—The diagnosis must mainly be determined by the history of the case, and by the facts that the symptoms come on suddenly and advance rapidly.

Often there are great difficulties experienced in arriving at a satisfactory opinion relative to the diagnosis, and I quote the following case not only because of its interest, but because it illustrates the scientific acumen of a distinguished member of the medical profession. Under the designation of "case of spinal apoplexy," Dr. Robert Jackson[2] says:

"On Sunday, May 2, 1869, Miss F. L., a bright, merry, healthy, and well-developed young lady, aged fourteen, arose as usual, but wh dressing said 'her fingers felt weak.' She, however, went to chur both morning and evening, and seemed quite well.

---

[1] "Traité théorique et pratique des maladies de l'appareil nerveux," Paris, 1869, tome ii., p. 423.

[2] Quoted in *Quarterly Journal of Psychological Medicine and Medical Jurisprudence*, New York, 1869, vol. iii., p. 810, from the *Lancet*.

"On Monday, she again got up as usual, but complained of the sa 'weak feeling' in her hands. Otherwise, she felt very well; particip in the usual studies of the day; and in the evening had a warm b enjoyed it, and got into it 'with the use of all her limbs.'

"On Tuesday she was much the same; ate a good breakfast, feeding herself, etc. During the forenoon, however, the weak feeling considerably increased, and I was sent for. I found her lying on her back in bed, quite merry, laughing, free from all pain, and rather amused, than otherwise, at her condition. She was, however, unable to shake hands with me, or to move her arms, except at the wrists; and failed altogether to pick up a pin placed on a book before her.

"On Wednesday, there was no material alteration. I observed, however, that the intercostal muscles were not acting quite freely; she seemed, too, to lie *heavier* in her bed, and she evidently was now unable to turn herself round. There was also a moist crepitant *râle* over all the chest, with a little cough. The secretions continued free, the pulse regular; and she ate, being fed, a good dinner of roast-beef.

"On Thursday, Sir William Jenner kindly saw her with me. Her general condition was not greatly altered; every sensation perfect; no anæsthesia; and she displayed her usual quick perception and intelligence. A careful examination, however, at this time, clearly demonstrated a great and decided loss of power in all the voluntary muscles of respiration, and in those muscles of the arms, back, and chest, supplied by the branches of the cervical nerves. The diaphragm, too, was becoming fixed, and there was slight lividity about the cheeks, with a fall of the natural temperature.

"From these symptoms, it became evident that there was some serious spinal lesion, implicating probably, and more particularly, the anterior branches of the cervical nerves, and the origin of the phrenics. Sir William Jenner diagnosed, and, as will be seen, with perfect accuracy, a clot in the cervical portion of the spinal cord, and he prognosed, notwithstanding the bright life and still merry laugh, a speedy and fatal result. This took place thirty hours afterward, without pain, without loss of consciousness or sensation, but only as the cessation of the power of respiration became more and more determined, with a desire to be raised 'higher and higher.'

"In this interesting case a post-mortem examination was kindly allowed, and made forty hours after death. There was slight opacity of the dura mater in several places. Brain congested and soft. A softened spot and ill-defined clot in the cerebellum. The whole cervical portion of the spine, but particularly anteriorly, and to the left side, was imbedded in an oblong clot of dark venous blood outside the membranes. The whole length of the cervical portion of the canal and the dura mater were deeply tinged by the color of the clot. The cervical nerves all passed through this effused blood, the inter-vertebral canals on both sides being filled with it. So soon as the seventh cervical vertebra was reached, the clot ceased, and the cord and canal assumed their normal condition and color. There was also a good deal of semi-clotted blood about the pons and the nerves arising from it.

"It is certainly a matter of much difficulty to account satisfactorily for this great effusion of venous blood in a subject so young and so apparently healthy and robust. No outward cause could be assigned; there had been no blow or injury; no illness ; no interrupted function ; but, living with kind and affectionate relations, she enjoyed every comfort and happiness. It might have been assumed that so great a lesion, situated in so important and vital a position, would have given rise to more decided and grave symptoms from the beginning. The only probable explanation is, that the effusion took place very gradually, had room to extend itself, and coagulated slowly, and imperfectly. Until the paralysis of the diaphragm showing dangerous interference with the functions of the phrenic nerves, nearly every symptom might have been attributed to one or other of those obscure forms of hysteria so frequently met with in practice."

Prognosis.—Death is the almost invariable result. I have, however, known two instances of recovery. In one of these the patient, a boy of about fifteen, was thrown from his horse. Paralysis supervened immediately, and there was a severe pain at about the eleventh dorsal vertebra. The bladder was also paralyzed. For several weeks his life was despaired of, but he eventually recovered with the paraplegia remaining, and the necessity of drawing off the urine with a catheter. I saw him five years after the injury. He was still paraplegic, and the bladder was still paralyzed. Careful examination failed to show any displacement or fracture of the vertebra, and I therefore felt warranted in concluding that there had been a spinal hæmorrhage, probably meningeal. The other case has been already cited. In this, the patient fell through a hatchway a distance of thirty feet, and struck on his back. Paralysis was almost immediate. He came under my care fifteen years after the event, and I diagnosticated a meningeal spinal hæmorrhage from the facts that there had been violent jerkings of the limbs and intense lumbar pain. There were no signs of fracture or displacement.

But in these, as in the following case, which I select from others similar, cited by Mr. Le Gros Clark,[1] there is, of course, room for doubt relative to the correctness of the diagnosis.

---

[1] "Lectures on the Principles of Surgical Diagnosis, especially in Relation to Shock and Visceral Lesions, delivered before the Royal College of Surgeons of England," London, 1870, p. 146.

The patient, a man thirty-six years of age, weighing 11½ ston gave the following account of himself: He was tripped up in the ro and fell heavily on his left hip, and then turned over on his back. trying to rise, he failed, not having any power of movement in eit lower extremity. He was at once brought to the hospital. On ad tance, he complained of pain in the lumbar region, and there was sli tenderness on pressing the spinous ridge of this part; but car examination failed to detect any irregularity, or any sign of mechanical injury of the vertebral column. There was entire loss of power in his lower limbs—he could not even move a toe; sensation was impaired; he said his limbs were numbed. There was slight priapism, and he was unable to micturate. His pulse was 60; but there were no signs of well-marked collapse. On the third day he was able to move his toes a little. On the ninth day, sensation was perfect; but he had made very little progress in regaining muscular power. Nearly three weeks elapsed before he was able to dispense with the catheter; and, at the expiration of five weeks, he was still almost as helpless in moving any part of his lower extremities. He remained in the hospital for four months, his health being tolerably good throughout. He was then able to get about very fairly, but with a shuffling, unsteady gait."

Mr. Clark then remarks:

"The causative accident in this case was slight, too trivial to produce fracture, and the symptoms were not those of sprain. There were no physical signs of displacement; yet, the paraplegia was marked; but not including corresponding loss of sensation, which would have been present if a displaced vertebra had pressed upon the cord. The slow recovery was a gradual confirmation of the diagnosis, that fracture with displacement was not the injury to which the symptoms were due.

"But, I must admit, I cannot dismiss from my mind that in these and similar protracted cases, there is something more than simple concussion needed to account for the duration of the symptoms; probably extravasation of blood into the theca or canal which is slowly absorbed. I do not think that the unequal effects produced on the several columns of motion and sensation forbid this supposition, for this effect is by no means uncommon, being usually in favor of sensation, where the inequality is noticed, and indicating that the anterior half of the cord and part of the lateral columns are the parts implicated. A child three and a half years old was admitted under my care, who had been run over by a heavy sand-cart, sixteen days previously; the wheel passed over the loins. There was nothing particular noticed at the time, except her inability to walk as well as usual. This inability increased, and, when seen by me, she could scarcely manage, when held up, to shuffle her feet along. She complained of no pain, had no difficulty in passing her water, and the sensibility of the legs seemed to be in no degree impaired. On careful examination, there was nothing abnormal to be observed in any part of the spine. The treatment consisted in rest, and friction of the back with liniment of ammonia. She remained in the hospital five weeks, and, then left quite well.

"A remarkable case was mentioned to me by the late Dr. Dyer, who acted for the Brighton Railway Company. A man was injured in a collision in the tunnel four or five miles from Brighton. He walked this distance with some difficulty into the town, and within twenty-four hours became entirely paraplegic. He recovered slowly, and after the lapse of two years was able to walk as well as before the accident. One spot on the back was always tender, and continues so still at times. The analogy between this case and a similar but fatal injury which I have already mentioned, seems to point to hæmorrhage as the probable cause of the protracted symptoms."

Of like character appears to have been the following case, which I cite from Dr. John Ashurst's[1] admirable monograph: "A male child of two years was admitted to the Pennsylvania Hospital on November 13, 1861, having a short time previously received a severe blow upon the back. There were no external marks of injury, but the lower limbs were paralyzed, doubling up upon themselves when an effort was made to place the child in an erect position. He was discharged cured, after two months, his treatment having consisted in little else than rest in a recumbent posture."

Several of the cases given by Mr. Erichsen,[2] in his excellent little work, appear to present many of the features of spinal hæmorrhage. It is, therefore, quite probable, making all due allowance for uncertainty in the diagnosis, that the affection in question, especially when resulting from traumatism, is not an entirely hopeless condition.

Morbid Anatomy and Pathology.—The blood in hæmorrhage of the spinal cord is effused either into the substance of the cord or into its membranes. It may, therefore, be situated in the nervous tissue; in the subarachnoidal space; in the intra-arachnoidal space; or in the space between the dura mater and the walls of the vertebral canal. In the first-named situation the gray matter is invariably—so far as our knowledge extends—the place of origination, unless we except certain possible traumatic cases. The clot shows a greater tendency to extend in the course of the long axis of the cord then laterally, and may vary in length from half an inch, or less, to three or four inches, or may involve the whole of the central portion of the cord.[3] The white substance rarely gives way to the interior pressure, but remains as a distinct boundary to the further extension of the clot in a lateral direction. Occasionally, however, this tissue gives way, and the clot appears as a tumor under the meninges. This was the case in a patient whose clinical history is related by Cruveilhier, and which is further remarkable by the fact that five years before the attack which terminated in death, the patient had experienced suddenly a severe pain in the neck, and paralysis of the left arm and leg. He recovered in three months. The post-mortem examination revealed the existence of an old apoplectic cyst in addition to the extravasation of the final hæmorrhage, which latter extended throughout the whole length of the cord, and had in several places broken through the white substance, being only restrained by the spinal membranes.

---

[1] "Injuries of the Spine, with an Analysis of nearly Four Hundred Cases," Philadelphia, 1867, p. 8.

[2] "On Concussion of the Spine, Nervous Shock, and other Obscure Injuries of the Nervous System in their Clinical and Medico-Legal Aspects," London, 1875.

[3] Hayem, *op. cit.*, p. 152; Cruveilhier, "Anatomie pathologique," book iii., plate vi

The clot may either present the general appearance of blood, and may, in fact, consist almost entirely of this substance, or it may, as is the case in cerebral hæmorrhage, consist of blood and the *débris* of the nervous tissue. The changes which ensue in the clot and in the limiting tissue are similar to those which take place in the brain under like circumstances.

In spinal meningeal hæmorrhage the blood is, as above stated, extravasated between the bones and the dura mater—extra-meningeal hæmorrhage; between the layers of the arachnoid—intra-arachnoidal hæmorrhage; or between the arachnoid and the pia mater—sub-arachnoidal hæmorrhage:

The *extra-meningeal hæmorrhages* are those which are especially apt to occur as the result of traumatic cause. The extravasation is generally extensive, and may occupy the entire extra-meningeal space—though, generally, it is circumscribed within much smaller limits. The cervical region is most apt to be its seat, and the dorsal next.

In *intra-arachnoidal* spinal hæmorrhage the blood is not effused in such large quantity as in the variety just described, and, moreover, generally has its source in a cerebral hæmorrhage—rarely being autocthonous. It is collected in a sac, and may exercise more or less compression on the cord according to the amount extravasated.

Sub-arachnoidal hæmorrhage is the rarest of all the forms. The blood is here extravasated into the meshes of the pia mater, and may compress the cord.

Of fifty-eight cases of spinal-meningeal hæmorrhage, cited by Hayem, thirty-eight were instances in which the blood was extravasated between the bones and the dura mater; eleven were intra-arachnoidal; and eight were sub-arachnoidal.

The symptoms which follow spinal hæmorrhage are the results of excitation and compression—the hyperæsthesia and the spasms being due to the former, and the anæsthesia and motor paralysis to the latter.

Treatment.—There is nothing to do in cases of spinal hæmorrhage but to maintain the patient in as quiet a condition as possible, and to keep ice constantly applied to the vertebral column. If there is time, ergot might, I think, be administered with advantage. In two cases which I have had the opportunity of observing from the first, both caused by falls from the loft of a stable, death took place within six hours; the symptoms gradually becoming more profound and advancing upward. After death, the hæmorrhage was found to occupy the whole length of the spinal canal, and was seated between the bones and the dura mater. Of course, in cases like these, no therapeutical means can avail, and, even in slighter cases, treatment is of little if any service.

We may, however, by perfect rest, ice to the spine, leeches to the anus, and the administration of ergot, sometimes prevent hæmorrhage in cases of injuries of the cord which otherwise might be followed by extravasation.

In cases not due to traumatism, and especially in those which are slow in their progress, more is to be expected from the use of remedial measures. Ergot should be energetically administered in large doses, two or three drachms every four hours, or, what is perhaps preferable, ergotin should be given to the extent of five grains hypodermically, as often. The other measures above mentioned should also be employed; with the view of causing absorption of the effused blood, the actual cautery applied to the spine in the vicinity of the lesion has been recommended. It should not be used till it is evident, from the non-progressive character of the symptoms, that the extravasation is no longer going on.

Strychnia is altogether inadmissible at any time in the course of the disease.

CHAPTER IV.

SPINAL MENINGITIS.

Inflammation of the membranes of the spinal cord may be either acute or chronic.

ACUTE SPINAL MENINGITIS.

Acute inflammation may be seated either in the dura mater, the arachnoid, or the pia mater of the cord, or may simultaneously attack all three membranes.

Symptoms.—The symptoms indicating inflammation of the dura mater are not very decided, and beyond the occurrence of pain may not be observed at all. When combined with inflammation of the arachnoid and pia mater, the phenomena are more pronounced.

Acute inflammation of the arachnoid does not of itself give rise to characteristic symptoms, and it is rarely the case that it exists separately.

Acute inflammation of the pia mater can, however, be recognized without difficulty. It begins with a chill, as do others of the phlegmasiæ, and this is soon followed by febrile excitement. At the same time there is intense pain in the back, which is aggravated by every movement of the patient, but not by pressure on the part of the spine over the diseased portion of the membrane. Those nerves which have their origins from the affected region are the seat of severe pain, which is transmitted through their trunks and branches to distant parts of the body. Spasms of the muscles of the back are commonly present. These are tonic in character, and may be so severe as to bend the body backward, producing an appearance like the opisthotonos of tetanus. At the same time the limbs below the seat of the lesion are strongly contracted. I have witnessed cases in which the knees were drawn up to the chin, and the heels to the buttocks.

At the same time there is impairment of motor power in all those parts of the body supplied by nerves coming from the cord below the diseased region, and in some cases voluntary control over the muscles is entirely lost.

While the affection is confined to the membranes of the lower portion of the cord, a fatal result may be deferred for some time, and the disease may become chronic; but, if it extends upward so as to involve the region from which the phrenic nerves arise, death very soon takes place by asphyxia.

So long as the spinal cord continues free from the disease, the reflex excitability and electro-muscular contractility remain unimpaired.

The bladder is not often involved, and the bowels may be obstinately constipated, or the fecal matters may be passed involuntarily.

CHRONIC SPINAL MENINGITIS.

This may arise in consequence of an acute attack, or it may be developed spontaneously. As in the acute form of the affection, pain constitutes a prominent feature, and is situated both in the spinal region and in other parts of the body. Spasms and contractions of the lower extremities, and spasms of the muscles of the back, are likewise prominent symptoms.

The pain in the spine is not increased by steady pressure over the vertebræ, but it is greatly aggravated by every movement of the body; for by such motion the nerves are compressed as they leave the spinal canal, and, as they are already in a condition of erethism, pressure cannot be borne.

The abnormalities of sensation are usually in the way of hyperæsthesia, which may sometimes be very acute.

The paralysis advances gradually, and rarely, at first, is very intense in any group of muscles. It is likewise subject to great variations in the degree of severity. Sometimes the patient finds that he walks tolerably well one day, while the next he can scarcely move a limb. These differences depend on the amount of fluid effused, which is subject to changes from day to day.

The bladder is sometimes paralyzed, the sphincter may be similarly affected, or this latter may be subject to repeated attacks of spasm, by which the evacuation of the urine is prevented.

The bowels, as in the acute form of the disease, may be either constipated, or the sphincter ani may be so paralyzed as to allow of the involuntary passage of the fecal matters.

Reflex excitability is rarely lessened, and is often considerably increased. In the case of a gentleman from Ohio who was recently under my charge for chronic spinal meningitis, the slightest touch on the sole of the foot was sufficient to cause the limb to be violently drawn up; and, in the case of a lady from New Orleans similarly affected, the contact of the bedclothes produced a like effect.

In several cases I have observed that any mental agitation, or even the attention directed to the affected limbs, was sufficient to cause violent spasmodic contractions.

Electro-muscular contractility is not generally impaired.

The symptoms are usually aggravated by the recumbent posture.

Bed-sores are a frequent accompaniment of chronic spinal meningitis.

Causes.—The most common cause of spinal meningitis, either acute or chronic, is exposure to cold and moisture. Several cases have come under my charge which clearly resulted from lying on the cold and damp earth, and from going to sleep in this situation. In one case which occurred in a railway conductor, the train of which he had charge was obstructed in its passage by a heavy drift of snow. While workmen were cutting a way through it, he lay down on a pile of snow, and, being greatly exhausted, quickly fell asleep. Soon after being awakened he had a slight chill and a mild fever, and the following day experienced severe pain in the back. This was soon followed by the other symptoms of spinal meningitis, not very intense in character, but persistent, for the affection passed into the chronic form. Two cases have come under my notice in which the disease was caused by the back being exposed to a strong and cold wind.

On account of this influence of cold in producing spinal meningitis, the disease is far more common in winter than in summer. Of the cases that I have treated wholly or in part during the last twelve years, by far the greater number occurred in the months from November to March, inclusive.

Exposure to the direct rays of the sun is said to induce spinal meningitis, but I have never witnessed a case in which this cause could reasonably be inferred. I may make the same remark in regard to the effects of strong muscular exercise.

It is, however, sometimes a consequence of wounds and injuries. Seven of the cases under my charge were due to traumatic causes.

Rheumatism is likewise an occasional, and syphilis quite a common cause.

Diagnosis.—The diagnostic phenomena of spinal meningitis, either of the acute or chronic form, are the pain in the back, increased on any movement of the spinal column; the pains in the course of the nerves having their origin from the diseased region; the tonic spasms of the muscles of the back, and of other parts of the body; the exaltation of reflex excitability; and the variations which take place in the extent and intensity of the paralysis.

Prognosis.—The course of spinal meningitis is generally progressively onward to a fatal termination—the patient dying either by the gradual extension of the disease upward so as to involve more important nerves in the lesion, by the development of some intercurrent affection, or by exhaustion. I have, however, seen five cases in which the disease was arrested, three of which will be more specifically referred to under the head of treatment. And Ollivier,[1] Brown-Séquard,[2] and Jaccoud,[3] admit the possibility of cure. When of syphilitic origin the prognosis is much more favorable.

Morbid Anatomy and Pathology.—The lesions found after death from spinal meningitis may be confined to any one of the membranes, but more generally are restricted to the pia mater and the sub-arachnoid space. They consist in thickening of the membrane, spots of opacity, turgidity of the vessels, and the effusion of a large quantity of spinal fluid. This fluid is occasionally clear, but is more frequently full of flocculent matter, or is tinged with blood.

The alterations found in the arachnoid are of similar character, with the addition that there are numerous hard cartilaginous plates scattered through the diseased part of the membrane, which vary in size from that of a grain of wheat to a mustard-seed.

The dura mater, when it has been the seat of inflammation, becomes thickened and adherent to the bone. Occasionally it is perforated by the supervention of gangrene, and the pus collected between it and the vertebræ escapes into the space between the dura mater and arachnoid, and excites general meningitis.

Ollivier reports[4] the case of a child three or four years old, who entered the hospital February 2, 1823. There were great difficulty of deglutition, a remarkable fixedness of the eyes, tetanic convulsions—trismus, opisthotonos—coma, and permanent contractions of the lower extremities. Death ensued on the twelfth day after admission. On post-mortem examination the membranes of the brain were found to be thickened and opaque, the substance of the organ was injected, and the ventricles contained an excessive amount of fluid. In the spine at the middle of the dorsal region, there was a very thick reddish infiltration in the cellular tissue, between the dura mater and the bony canal. On incising the membranes, it was seen that their cavity was filled with serum; the vessels of the pia mater were intensely congested. The substance of the cord was slightly injected.

---

[1] "Traité des maladies de la moëlle épinière," etc., Paris, 1827, tome ii., p. 295.

[2] *Op. cit.*, p. 302.

[3] *Op. cit.*, p. 82.

[4] "Traité des maladies de la moëlle épinière," troisième édition, Paris, 1837, tome ii., p. 272.

Michaud,[1] under the name of *external pachymeningitis*, has described an inflammation of the dura mater, which he has found to be the affection of the membranes generally produced by Pott's disease. It consists in a thickening of the dura mater by deposits of yellow-colored granulations, which by their confluence form plates which are attached by their inferior surface to the membrane. Under them the dura mater appears to be healthy. At first they are only developed in the vicinity of the osseous lesion, but they have a tendency to extend, and may involve the whole length of the membrane. The existence of these formations was first noticed by E. Wagner[2] in a case of Pott's disease, of which he made the post-mortem examination. This inflammation of the dura mater may result in little abscesses, scattered through its lamina, or larger collections of pus may be formed in the substance of the new formation.

The symptoms, as Leyden[3] and Rosenthal[4] admit, are simply those of the other forms of spinal meningitis.

The internal surface of the dura mater may also be the seat of morbid processes. Two of these modes have been differentiated. One constitutes the *cervical hypertrophic pachymeningitis* of Charcot;[5] the other is the *internal hæmorrhagic pachymeningitis* of A. Meyer[6] and others. As described by Charcot, cervical hypertrophic pachymeningitis consists in an alteration of the meninges, especially the dura mater. The seat of the lesion is variable, but the cervical enlargement appears to be the place generally affected. The alteration of the dura mater is the primary fact; the other membranes, the cord itself, and the nerves coming from it, subsequently become involved. Formerly, the disease was mistaken, as by Laennec, Andral, and Hutin, for a primary

[1] "Sur la méningite et la myélite dans le mal vertebral," Paris, 1871, p. 9.

[2] "Pathologisches, anatomisches und klinisches Beiträge zur Kentniss der Gefassnerven," *Archiv der Heilkunde*, Heft 4, 1870, S. 321.

[3] "Klinik der Rückenmarks-krankheiten," Berlin, 1874, Erster Band, S. 388.

[4] "Klinik der Nerven-krankheiten," Stuttgart, 1875, S. 280.

[5] "Mémoires de la Société de Biologie," 1871, p. 35, and "Leçons sur les maladies du système nerveux," Paris, 1874, p. 246.

[6] "De Pachymeningitide cerebro-spinali interna," Bonnæ, 1861.

affection of the spinal cord, and was described by them as hypertrophy of this organ; and the error is in a measure sustained by the fact that in cases of the disease in which the vertebral canal is opened, the spinal cord and its membranes are seen to completely fill the canal.

But, upon making a transverse section of the cord, it is at once perceived that the swelling is due to the thickening of the envelopes, and that the marrow, so far from being enlarged, is in reality compressed and flattened from before backward.

In the accompanying engraving (Fig. 35) taken from Joffroy's[1] memoir, the appearances are well exhibited (*a*, the hypertrophied dura mater; *b*, nerve-roots traversing the thickened membranes; *c*, the pia mater confounded with the dura mater; *d*, lesions of chronic myelitis; *e*, section of canals newly formed in the gray substance).

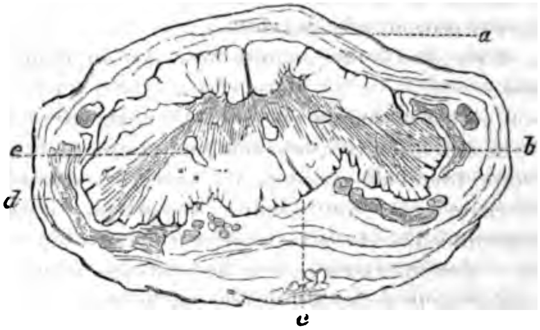


FIG. 35.

As will readily be perceived, the pia mater is involved in the morbid process, but not to the same extent as the dura mater. This latter, when carefully examined, is seen to be composed of numerous concentric layers, and is adherent on the outside to the vertebral ligament, and on the inside to the pia mater.

Sometimes the thickened, hypertrophied membrane seems to be constituted of two layers; the one external, the other internal. This last, which appears to be a new formation, consists of a dense fibroid tissue. It is, therefore, quite distinct from those soft and very vascular neo-membranes which, in the spinal as well as in the cerebral dura mater give rise to hæmatoma—constituting in the former the internal hæmorrhagic pachymeningitis to be presently described.

The spinal cord itself participates in the alteration, which has all the characteristics of a transverse, irregularly disseminated myelitis, attacking as well the central gray matter as the white columns.

The peripheric nerves are affected by the spinal lesion, both in their radicles within the cord, and in their trunks, as they pass through the thickened and inflamed membranes. The anterior and posterior roots are about equally involved, and hence, as symptoms, there are both derangements of motion and of sensibility.

---

[1] "De la pachyméningite cervicale hypertrophique," Thèse de Paris, 1873.

The symptoms, as given by Charcot, are, in the first place, extremely violent pains, which occupy mainly the posterior part of the neck, but which extend to the top of the head and to the superior extremities. These pains are accompanied with rigidity, especially marked in the neck, which is immobile, as in Pott's disease, occupying the sub-occipital region. They are generally quite constant, but are more violent at some times than at others. They extend to the joints, which, however, are not ordinarily the seat of swelling, and with these pains there are the various sensations of numbness in the superior extremities, and some degree of paralysis. Sometimes there are bulbous and pemphigoid eruptions.

The second period is characterized by other symptoms, which appear to be due to the extension of the meningeal lesion to the spinal cord, and to a more profound alteration of the peripheric nerves.

The limbs cease to be painful, but they become paralyzed, and the muscles are atrophied, and the atrophy extends to all the muscles of the extremity. But, speaking only of the muscles of the arm and forearm, it is notable that those which receive their innervation from the ulnar[1] and median nerve are especially affected, while those which are supplied by the radial nerve almost entirely escape. From this peculiarity a certain character of deformity results, which, though met with in other diseases, and not always seen in the affection under notice, is, nevertheless, not a feature of other forms of muscular atrophy. It is, consequently, a diagnostic mark of some value (Fig. 36). To these

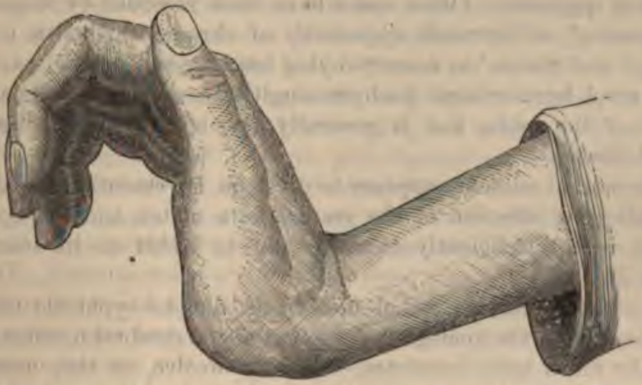


FIG. 36.

---

[1] M. Charcot says, *du nerf radial et du nerf médian*, but it is evident from the context, as well as from what follows, that *radial* is a misprint for *cubital* (*op. cit.*, p. 251) The cut also shows the error.

symptoms are added contractions, and often anæsthesia, which may tend from the extremities to the trunk. After a while, the infe[illegible] extremities become paralyzed, and eventually contractions ensue[illegible] them also.

Charcot does not regard hypertrophic pachymeningitis as a ne[illegible]sarily incurable affection: for a woman, who, for five or six years, exhibited all its characteristic symptoms, being confined to her bed for a long period, recovered so far as to be able to walk and to use her hands in some labors.

Internal hæmorrhagic pachymeningitis is, in the spinal canal, the analogue of cerebral pachymeningitis or hæmatoma of the dura mater. Its differentiation from spinal meningitis was first made by Albers, though he failed to indicate its characteristic features. A. Meyer[1] first pointed out its essential nature. An officer had for some time been subject to vertigo, accessions of heat, and arterial throbbings, in the head and back. He recovered, but was subsequently seized with paralysis, mental derangement, incontinence of urine, and agonizing pains in the head. When these symptoms had lasted, with gradually-increasing intensity, for about a year, he died. The autopsy revealed the existence of cerebral meningitis, and of a false membrane attached to the cranial dura mater, and to the same membrane in the vertebral canal as far down as the last dorsal vertebra. This membrane was fibrous, and composed of several laminæ, between which were extravasations and masses of pigment. Other cases have been recorded by Magnan and Bouchereau,[2] as the result apparently of chronic alcoholism; and by Charpy[3] and Simon,[4] as accompanying insanity and general paralysis.

Internal hæmorrhagic pachymeningitis is rarely unaccompanied by lesions of the brain, and is generally associated with the like intracranial disease.

The morbid anatomy appears to differ in no essential respect from the analogous affection of the cranial dura mater, and the symptomatology is not sufficiently characteristic to admit of its recognition during life.

In cases of chronic spinal meningitis, due to syphilitic taint, the symptoms, as in the analogous condition of the cerebral membranes, are generally much more restricted, and may involve, as they usually do, the meninges in relation with the antero-lateral columns only. In such cases the lesion is presumably circumscribed, and the gummy exudation is likewise limited. The symptoms then relate almost entirely to the power of motion, either of one or both lower extremities, and there is thus more or less extensive paraplegia. The lower dorsal and upper lumbar regions are, in my experience, almost the only parts of the cord attacked in such cases; though I have occasionally witnessed instances in which the lesions were multiple, some of them being high up in the cord.

---

[1] *Op. cit.*

[2] "Mémoires de la Société de Biologie," 1869.

[3] Cited from unpublished notes, by Hayem, *op. cit.*, p. 90.

[4] "Ueber den Zustand des Rückenmarks in der Dementia paralytica," *Griesinger's Archiv*, Heft 7 u. 2.

In an interesting case, the details of which are very fully given by Jaccoud,[1] a man was paralyzed in both lower extremities. When the patient came under observation, the paraplegia had already lasted two months, and had been fully developed in three days: standing was impossible, the right leg was more paralyzed than the left; there were neither contractions nor atrophy; there were no involuntary movements; the motility of the trunk and superior extremities was perfect; electro-muscular excitability was not impaired; tactile sensibility and sensibility to pain and heat were normal in the right inferior extremity; but in the left, though tactile sensibility was good, the sensibility to pain and heat was impaired; indeed, the sensation to impressions, ordinarily painful, was entirely lost throughout the whole extent of the limb.

In regard further to the case of which I have given but a brief abstract, M. Jaccoud remarks:

"The nature of the lesion can be very easily determined. The paraplegia was developed in three days, in an apparently healthy man. There had been no fever, pains, sensation of constriction, convulsions, or contractions. The lesion was very limited, the portion of the cord situated above was not altered, and the morbid process involved one side more than the other. I know of no condition which fulfills all these phenomena but compression of the cord. Paraplegia of rapid invasion is observed, it is true, in acute myelitis, in acute meningitis, in spinal hæmorrhage, in meningeal spinal congestion, and in hydrorachis; but it is then accompanied with fever or pain, symptoms which have been entirely absent in our patient. Moreover, these lesions do not produce so limited a disorder as that before us. The compression of the cord in this case is from before backward, and the anterior columns are more affected than the posterior; but on the right side the compression has involved the gray sensory elements of the posterior spinal system, leading to loss of thermic and painful sensibility in the left lower extremity.

"Such is the pathogenetic diagnosis of our paraplegic patient; it is founded entirely on the physiological interpretation of the symptoms; and, as the case is very strongly marked, analysis permits us to notice all the particularities of the lesion of the cord. You can ask nothing more complete; it is a physiological diagnosis *par excellence*; it is perfect. Here, gentlemen, appears the superiority of medical over physiological diagnosis; and I am happy that this occasion permits me to insist upon your appreciation of this capital truth, that others have vainly opposed. What does this very exact physiological diagnosis teach us in regard to the prognosis? Nothing, absolutely nothing; it is a dead letter. Our patient has a compression of the cord at the tenth dorsal vertebra. This compression is stronger on the right than on the left side; it has interrupted the conductibility of the motor columns of the cord on both sides, and of the sensory columns on the right side, but it has not interfered with the posterior white columns, or the two orders of nerve-roots. But, knowing all this, are we the better enabled to be of use to our patient, who cares only for one thing, and that is the recovery of the use of his legs? No! a thousand times no! These scientific data, acquired with such labor, are sterile, and our physiological diagnosis is powerless to help us. It teaches us nothing relative to the probable issue of the disease; nothing touching the treatment to be employed. But let us substitute the physician for the physiologist; let us bring the principles of clinical diagnosis to bear upon the subject, and we shall discover something of the future of the patient, and how we are to treat him. This man is syphilitic, and that one word, which is the index of the medical diagnosis, at the same time points out to us the nature of the compression which the cord is undergoing, reveals the prognosis of the paraplegia, and directs us as to the treatment to be employed.

---

[1] "Leçons de clinique médicale faites à l'Hôpital de la Charité," deuxième édition, Paris, 1869, p. 446.

"This patient has, up to this time, had none of the accidents called secondary; he certainly has not yet reached the tertiary stage; at most we can only say that he has arrived at that transition stage which often separates superficial from profound syphilitic determinations. He presents no visible lesion of the bones, and in that situation we could only allege an exostosis or a vertebral periostitis as a cause of the compression of the cord. I am more inclined to believe that the lesion is one of those meningeal affections of the kind described by Knorre,[1] consisting in circumscribed exudations, which may remain latent if they are very small, but which, if large, may cause compression and consequent paraplegia."

The patient ultimately recovered, under the use of the bichloride of mercury and the iodide of potassium.

A case, very nearly identical in its chief features with that of M. Jaccoud, came under my care a few months since, in consultation with Dr. Van Wyck, of this city, in which recovery was complete under like treatment.

In relation to these exudations of the spinal membranes, Virchow[2] declares that little is known of their morbid anatomy; and Charcot[3] asserts that they are not common—basing his opinion, however, only on the small number of post-mortem examinations which have been made, in which these formations have been found. A parity of reasoning would throw hysteria, for instance, out of nosology altogether. We are not likely to have much information in regard to the morbid anatomy of so curable a disease as syphilitic spinal meningitis. So far as our information extends, the condition induced in the spinal membranes by syphilitis does not differ essentially from that caused by the same influence in the cerebral membranes, and which has been fully considered in the present work under the head of basilar cerebral meningitis. In this view I am supported by Buzzard,[1] Lagneau,[2] Gros and Lancereaux,[3] Zambaco,[4] and others.

---

[1] "Ueber syphilitische Lähmungen," *Deutsche Klinik*, 1849.

[2] "Pathologie des tumeurs," Paris, 1869, tome ii., p. 454.

[3] "Leçons sur les maladies du système nerveux," deuxième partie, second fascicule, p. 80.

The theory of the symptoms observed in spinal meningitis is, that they are due to two immediate causes, excitation and pressure. The former is the result of the hyperæmia, the latter of the exudation, or of the increased amount of spinal fluid causing pressure.

Treatment.—In the acute form of spinal meningitis, active measures are required. The application of leeches to the painful part of the spine, or of cups, so as to effect local depletion, will generally prove useful. Hydragogue cathartics are also beneficial, for by their action the vessels of the inflamed membranes are depleted of their blood, and the excessive amount of spinal fluid effused is in consequence more readily absorbed.

Mercury may also be advantageously administered either by inunctions with mercurial ointment or by calomel given internally, or by both these means. Calomel should be given in doses of from one to two grains every three or four hours, till the system is brought under its influence, as manifested by fetor of the breath.

The patient should be kept as quiet as possible, and should be enjoined not to lie on the back. For the relief of the dorsal and other pains, suppositories, containing each, half a grain of codeine, are often efficacious. They may be administered night and morning.

In the chronic form of the disease, depletion by bloodletting in any form is not so beneficial as in the acute variety or as in spinal congestion. Blisters are more admissible, and scarcely ever fail to do good. They should be applied on each side of the spinal column near the diseased region of the cord, and as soon as one heals another should take its place. Purgatives are also useful for the same reasons which prevail in acute spinal meningitis.

Iodide of potassium is always a valuable agent, indeed more so than any other remedy employed in chronic spinal meningitis. I employ it in the form of a saturated solution, which contains about a grain to each drop. Of this, I administer the first day seven drops three times, preferably before meals; the next day eight drops to the dose, the next nine, and so on, till the patient takes from forty to sixty drops at the dose, according to circumstances. The iodide of potassium always acts best when largely diluted with water, so that, as the doses are increased, an additional quantity of water should be used.

---

[1] "Clinical Aspects of Syphilitic Nervous Affections," London, 1874, p. 70.

[2] "Maladies syphilitiques du système nerveux," Paris, 1860.

[3] "Des affections nerveuses syphilitiques," Paris, 1861.

[4] Ibid., Paris, 1862.

I very often employ the corrosive chloride of mercury in combination with the iodide of potassium, in doses of the sixteenth of a grain with each dose of the iodide.

The treatment with iodide of potassium and mercury is still more strongly indicated in those cases which are of syphilitic origin.

Diuretics may also frequently be given with advantage. Their object is the same as that which governs in the administration of purgatives.

In two of the cases cured, to which reference has been made, I derived the greatest benefit from repeated blisters, and the persistent use of iodide of potassium. The latter was carried to the extent of fifty grains three times a day in one of these cases, and sixty-five in the other.

At the same time the primary galvanic current was applied to the spine in the manner recommended for spinal congestion, and the induced current to the paralyzed limbs. I am very sure that electricity in both these forms should be used in most cases of chronic spinal meningitis. The following case, reported by J. Frank,[1] and quoted by Ollivier,[2] of acute spinal meningitis, is instructive:

"A captain, aged forty-two years, of sanguineo-bilious temperament, subject to rheumatic pains and hæmorrhoids, and addicted to the use of alcoholic liquors, was suddenly seized on the evening of the 2d of March, 1819, with a chill, which was soon succeeded by a burning fever, accompanied by pain in the lumbar region. During the night the pain increased, extended as high up as the occipital region, and gradually acquired great intensity. J. Frank was called in the morning at five o'clock, to see the patient, who was suffering acutely. He was uttering loud groans, was lying on his belly, with the superior and inferior extremities stretched out to their full length. To the questions put to him, the patient answered with great difficulty that he had pains all over his body, that he was unable to open his eyes, that his teeth were strongly clinched, and that a burning and pulsating pain extended from the occiput to the lower extremity of the vertebral column. The limbs, especially the inferior, were without sensation, but were agitated by occasional jerkings. There was such a constriction of the chest that breathing was scarcely possible, and the abdomen was likewise in a state of contraction. There were constipation, incontinence of urine, a pulse soft but 100 per minute, occasional palpitations of the heart, and a hot and dry skin.

---

[1] "Praxeos Med., etc., de rachialgite," tome vi., p. 76, Turin, 1822.

[2] *Op. cit.*, p. 295.

"Frank at once opened a vein in the foot, and abstracted sixteen ounces of blood. A dozen leeches were applied around the occiput, and as many scarified cups on each side of the spine. A decoction of tamarinds was given as a cathartic. These means were sufficient to restore the health of the patient in a few days. The bloodletting produced an almost immediate cessation of all the symptoms; for, a short time after its employment, the movement of the eyelids became easy, as well as that of the jaw; sensation reappeared in the extremities, and the dorsal pain diminished considerably in intensity."

As Ollivier remarks in regard to this case, several of the symptoms are those of spinal congestion. The sudden supervention of the disease, as well as its rapid disappearance, points to that affection. Nevertheless, its general features are those of acute spinal meningitis—an affection which, of course, cannot exist without congestion.

In a very interesting case under my charge several months ago, a cure of the spinal disorder, which was chronic spinal meningitis probably of syphilitic origin, was accomplished by the use of the iodide of potassium and the corrosive chloride of mercury, as recommended on pages 449 and 450. In this case the affection had lasted for several years, and extended from the occiput to the lower extremity of the spinal cord. The limbs were constantly subject to violent spasmodic jerkings, and both legs and one arm were in a permanent state of contraction, which had existed for three years. Under the use of the iodide and the mercury, as mentioned, the pain, which had been intense, ceased, the spasms of the limbs were stopped, the bladder regained its expulsive power, the bowels again began to act without purgatives or injections being required, and the limbs could be moved as extensively as the rigid contractions permitted. These had existed so long that the flexor muscles had become much shortened, and the skin in the groins and popliteal spaces was tense and unyielding. The accompanying woodcut (Fig. 37) shows the positions of the legs and arm at this time. Under these circumstances I requested the advice of my friend Prof. L. A. Sayre, and after consultation it was determined to divide the tendons of the tensor vaginæ femoris, the sartorius, the gracilis, and the biceps, on each side. When this was done by Prof. Sayre, the patient being under chloroform, careful but powerful efforts at extension were made, and the skin in the popliteal space on both sides was necessarily torn, owing to its contraction and inelasticity; the limbs were thus brought into a state of complete extension, and, by a system of weights and pulleys similar to that used in Buck's fracture apparatus, they were kept in this position. The patient was, however, too weak to endure the fatigue of the necessary extension and confinement. He

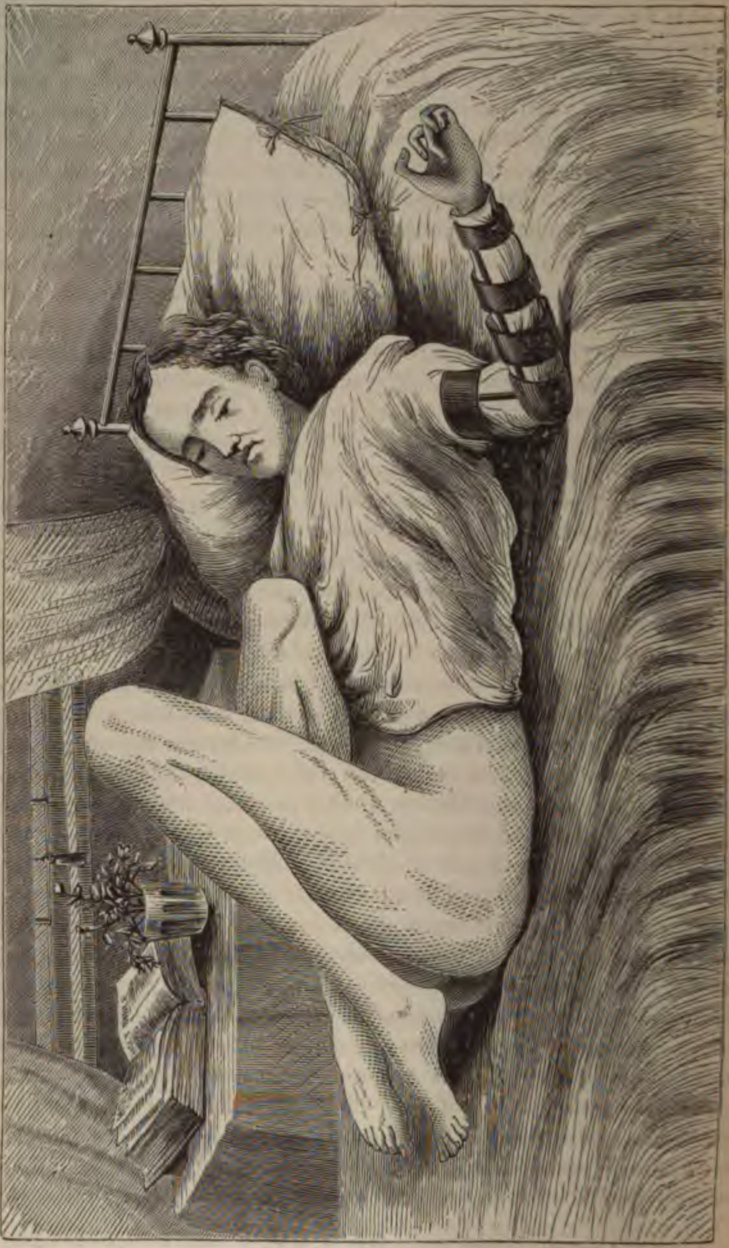


Fig. 37.

took off the weights whenever they caused pain or great uneasiness. To add to the difficulties, a large bed-sore formed on the right buttock, and the strength of the patient declined so rapidly that, in order to save his life, the apparatus had to be entirely removed. He rapidly recovered, but, as cicatrization went on, the limbs again became contracted, and in the course of two or three months were as bad as ever. Pain in the back soon afterward supervened, the legs and one arm began to be affected with spasms, and the paralysis also returned. A renewal of the former medication again caused relief, and the patient has to this day remained free from any spinal disease, though his legs are still contracted. This is the third case of cure referred to as happening in my experience.

For the cure of the bed-sores the method recommended by Dr. Brown-Séquard may be used. It consists in the alternate application of sponges, one of which is saturated with hot water and the other with cold water. This should be done for five or ten minutes every day, and the effect is to increase the activity of the circulation of the part, and to promote the formation of granulations.

But I have generally preferred the method by galvanism first suggested and employed by Crussel,[1] of St. Petersburg, and which I used for the treatment of indolent ulcers with almost invariable success, in 1859, when surgeon to the Baltimore Infirmary. The method was also recommended by Mr. Spencer Wells.[2] During the last twelve years I have employed it to a great extent in the treatment of bed-sores caused by diseases of the spinal cord, and with scarcely a failure—indeed, I may say without any failure except in two cases where deep sinuses had formed which could not be reached by the apparatus.

A thin silver plate, no thicker than a sheet of paper, is cut to the exact size and shape of the bed-sore. A zinc plate of about the same size is connected with the silver plate by a fine silver or copper wire six or eight inches in length. The silver plate is then placed in immediate contact with the bed-sore, and the zinc plate on some part of the skin above—a piece of chamois-skin, soaked in vinegar, intervening. This must be kept moist, or there is little or no action of the battery. Within a few hours the effect is perceptible, and in a day or two the cure is complete in the great majority of cases. In a few instances a longer time is required. I have frequently seen bed-sores three or four inches in diameter, and half an inch deep, heal entirely over in forty-eight hours. Mr. Spencer Wells states that he has often witnessed large ulcers covered with granulations within twenty-four hours, and completely filled up and cicatrization begun in forty-eight hours.

---

[1] *Neue Med.-Chirurg. Zeitung*, No. 7, 1847, p. 235.

[2] "Lectures on Electricity and Galvanism," by Dr. Golding Bird, London, 1849, appendix. There is an American edition of this very interesting little book, but it has long been out of print.

During his recent visit to this country I informed him of my experience, and he reiterated his opinion that it was the best of all methods for treating ulcers of indolent character and bed-sores.

Ergot is not so generally useful as in congestion, though I rarely fail to give it at some time or other in cases of chronic meningitis, with a view to the relief of the accompanying congestion. Strychnia is not at all admissible at any time. Reeves[1] recommends it in those cases in which pains, cramps, and contractions, are absent, but I have never seen such cases. Indeed, a case in which they were not prominent symptoms could scarcely be regarded as one of spinal meningitis.

In the meningitis and myelitis resulting from Pott's disease, the actual cautery is of inestimable value. Its efficacy has been very strongly insisted on by Charcot and Michaud, both of whom give cases in illustration of its value. Within the last year I have treated five cases of this complication with the agent in question, and with beneficial results in all. It is an error to suppose that the paraplegia so generally attendant in the vertebral disease is the result of the compression of the cord. For there may be paraplegia—as I had the opportunity of seeing quite recently in a case of Pott's disease under the care of Dr. F. D. Lente, of Cold Spring, and in which I was consulted, when there is no deformity whatever; and the paraplegia may disappear, the curvature remaining undiminished. This was the case in a patient sent to me by Dr. Butler, of Baltimore, who had been the subject of Pott's disease several years previously, and who had recovered, with very great curvature, but without paralysis. A few weeks, however, before coming under my observation, the paraplegia had returned, the curvature remaining the same. Cruveilhier, as Charcot reminds us, pointed out, long ago, the fact that the spine may be the seat of the most extraordinary deformities without the cord being compressed.

In a lecture delivered at the Salpêtrière, M. Charcot[2] related the case of a young Polish girl affected with Pott's disease, complicated with paraplegia, who left Warsaw to consult him, but who, on her way to Paris, stopped in Berlin, to obtain Langenbeck's advice. The great German surgeon counseled her not to allow the cautery to be used, but she, nevertheless, proceeded to Paris. After the second cauterization she walked, and fifteen days subsequently she paid a second visit to Langenbeck, "furnishing to him," as the reporter remarks, "an irrefragable proof that empiricism is a good thing, when it is accepted and recommended by men of science."

The number of cauterizations need not exceed five or six, and they are best made with the disk-ending iron with platinum tip, which should be applied at several points on each side of the diseased vertebræ.

---

[1] "Diseases of the Spinal Cord and its Membranes, and the Various Forms of Paralysis arising therefrom," London, 1858, p. 55.

[2] *Gazette Médicale de Paris*, 5 Décembre, 1874.

CHAPTER V.

THE INFLAMMATIONS OF THE SPINAL CORD.

The subject of inflammation of the spinal cord has, within the last few years, been so greatly amplified in all its details by those eminent French pathologists, Charcot and Vulpian, and their pupils, and so much exact information has been obtained through their investigations, that the arrangement followed in the previous editions of this work, based on the morbid anatomy, as then known, no longer represents the actual state of the science. I shall, therefore, consider the inflammatory affections of the spinal cord according to a plan somewhat modified from the systematic table of Clément[1]—a table constructed from the most recent data furnished by the authorities above mentioned. In so doing, I shall omit those disorders which have only a theoretical existence, or which, in my opinion, have been, on insufficient data, assigned a definite patho-anatomical position.

I.

ACUTE MYELITIS.

a. Acute General Myelitis.

In acute general myelitis the whole extent of the cord is involved in the morbid process.

Symptoms.—The onset of the disease is sudden. A chill is generally the first symptom observed, and this is followed immediately by high febrile excitement, during which the pulse may be as frequent as 160 per minute. The temperature of the body is slightly elevated, but rarely reaches 103° Fahr. Alterations of sensibility and motility are noticed with the inception of the fever.

Among the first, pain in the back is prominent. This is usually most severe in the dorsal region, and is aggravated by percussion and by the passage of a sponge saturated with hot water, or one with cold water, over the affected region. It is not, however, so intense in character as that attendant on acute spinal meningitis, and it is not increased by movements of the limbs or of the vertebral column, in which respects it differs from the pain due to this last-named disease.

In addition, there are various derangements of the cutaneous sensibility in those parts of the skin below the seat of the disease. These consist of formication, "pins and needles," a sensation as if water were trickling over the skin, as if the limb were asleep, and of sensations of cold or heat. Anæsthesia is the most common general condition of the skin, and it is often accompanied with cutaneous pains, which are the more intense the more profound is the anæsthesia. Thus, if we have ascertained that the cutaneous sensibility is very much impaired at a particular spot, we will frequently find this spot the seat of severe and spontaneous pains. In such cases, too, a prick with a pin is felt, but the ability to distinguish the two points of the æsthesiometer is lost, even when they are widely separated. Indeed, they may not be felt at all unless they are so used as to cause pain. I have several times observed patients whose tactile sensibility was almost entirely gone, but whose sensibility to pain was so great that they could not endure the contact of the bedclothes. The distinction, therefore, between insensibility to touch—generally called anæsthesia—and insensibility to pain—analgesia—must be clearly made.

---

[1] "Note sur les myélites d'après les travaux français récents," Paris, 1875, p. 7.

A sensation of constriction around the body is sometimes experienced, and the limbs are likewise often the seat of a like symptom, giving the impression to the patient that they are encircled by tight cords or incased in closely-fitting armor.

Hyperæsthesia is occasionally present, but probably not unless there is meningitis associated with the myelitis.

Motility is affected at a very early period of the disease, and at first consists of simple twitchings of the muscles, and paralysis. The latter comes on with great rapidity, and may become complete in a few hours. Jaccoud[1] states that he has seen this result produced in thirty-six hours, and Ollivier[2] cites several cases to the same effect. The bladder is almost invariably paralyzed, as are also its sphincter and that of the anus. There is, therefore, dribbling of the urine, and the fæces are evacuated involuntarily as soon as they pass into the rectum.

Reflex excitability is entirely abolished in acute general myelitis. Tickling the sole of the foot, therefore, fails to produce any movement.

Electro-muscular contractility is diminished, unless, perhaps, in the very earliest stage of the affection, and there is always a tendency to rapid atrophy of the paralyzed muscles.

The temperature of the affected limbs begins to fall from the very first, and may be diminished by as much as 3° Fahr. Sloughs and bedsores generally make their appearance about the sixth day, though I have several times seen them form at a much earlier period. Although they usually occur in those parts of the body—as the sacrum, nates, and hips—which are subject to pressure, it is very certain that the decubitus is not their primary cause. In three instances I have had them result, in cases under my charge, within twenty-four hours after the inception of the disease.

Besides the foregoing symptoms, there are others referable to the viscera, and which may occur almost simultaneously, or in marked sequence, as the morbid process extends through the length of the cord. Thus, there may be frequent and almost constant painful erections, vomiting, derangement of the liver, irregular action of the heart, difficult respiration, and more or less impairment of the faculty of swallowing. The voice may be abolished, and the muscles of articulation be so far paralyzed as to render even a whisper impossible.

---

[1] *Op. cit.*, p. 318.

[2] *Op. cit.*, chap. huitième, "Myélite, ou inflammation de la moëlle épinière."

The urine is often, if not invariably, alkaline. This is not the result of contact with the mucus retained in the paralyzed bladder, for, if this organ be thoroughly cleansed with water, the urine collected from it with a catheter is found to be of alkaline reaction. This alkalinity is doubtless due to the presence of an excessive proportion of the ammonio-magnesian phosphates.

The quantity of urine is diminished, and albumen, pus, or blood, may be present in it, besides the large amount of vesical mucus which is so prominent a constituent.

Acute general myelitis ordinarily runs its course in about ten days, though it may terminate in death in a much shorter period, or be prolonged for several weeks. Death is due either to asphyxia or exhaustion. The former result is obtained when the inflammation reaches the upper cervical region, and the muscles of respiration become paralyzed, or the action of the heart greatly interfered with. In consequence of the paralysis of these muscles and of those concerned in deglutition, mucus accumulates in the air-passages and pharynx, and may lead to sudden asphyxia.

Such is a general view of the symptomatology of acute general myelitis, when the whole or greater part of the spinal cord is involved. It is not, however, to be supposed that the phenomena are all present at the same time. Such a condition very rarely exists. As a rule, the inflammation advances from below upward, and the symptoms occur in order as the morbid action progresses. Sometimes, however, the central region of the cord is first attacked, and the extension takes place in both directions.

b. Acute Partial Myelitis.

In this form of the affection the inflammation is restricted to a limited portion of the cord, and, as a consequence, the symptoms are less profound and extensive than in the general form. The morbid process may be confined to a very small part of the cord, or may involve the cervical, dorsal, or lumbar regions, with, of course, some variation in the symptoms, according to the situation.

Symptoms.—The pain in the back is similar to that experienced in the general form, and it is, like that, excited or aggravated by percussion or by the passage of a sponge saturated with very hot or cold water over the affected locality.

The aberrations of sensibility are less strongly marked, but, on account of the slower progress of the disease, they are of longer duration.

In a case now under my charge, and in which there is the pain in the spine, presenting the above-mentioned characteristics, the patient, a gentleman about thirty years of age, has, in addition to these marked symptoms, nearly absolute anæsthesia in the left lower extremity, which existed as the only phenomenon of any moment for three or four weeks.

The sense of constriction around the body is generally but not always present, and its seat marks the upper limit of the inflammation.

Another symptom, often noticed, is one to which Charcot has called special attention, and that is, the inability of the patient to localize his sensations. Sometimes this is surprisingly manifested. In a patient whom I brought at my clinique before the class of the University Medical College, a prick of a pin made in the right thigh was referred to the left thigh, and one made on the left foot was felt on the left knee. So far as the intensity of the sensation was concerned, it was as great as, if not greater than, in health. This is not an unusual circumstance. In a case cited by Charcot,[1] the sensibility to cold, to contact, and to tickling, was abolished, and yet, when the patient was pinched, an acute sensation of pain was experienced. This pain was accompanied by spinal symptoms:

1. There was error as regarded location: the leg was pinched, but the pain was referred to the hip, then to the opposite hip, and, finally, to the whole length of both limbs.
2. The sensation was assimilated to a vibration or a trembling.
3. It was the same for the different methods of excitation; it not only ensued on pinching, but on the application of cold.
4. It lasted during a quarter of an hour, and sometimes longer.
5. Sometimes it was not perceived till an appreciable interval of time had elapsed. In a case cited by Romberg, this interval was thirty seconds.[2]

As Charcot says, this delay in appreciating sensations is due to a profound lesion of the gray substance of the cord.

The paralysis of motion observed in partial acute myelitis is less extensive than that which exists in the general form of the disease. In the early stages there are convulsive movements in the muscles supplied by the nerves, having their origin in the affected portion of the cord; but, eventually, the loss of power becomes more or less complete. It may at no time, if the lesion be very circumscribed, extend beyond the point of slight diminution.

The reflex excitability is generally augmented. In a case now under my charge, in which the morbid process apparently only involves a segment of the cord in the lower dorsal region, the slightest touch of the lower extremities is followed by movements as intense as those in tetanus. Charcot, as stated by Dujardin-Beaumetz, arranges the phenomena under this head into two classes: that in which there is a simple exaggeration of the spinal excitability, and that in which there is the continuance of the spasms, which are, in my opinion, improperly designated spinal epilepsy. This spinal epilepsy may appear under two forms: either as tetaniform or tonic convulsions, or as saltatory cramps—clonic convulsions. In several cases I have witnessed both of these types in the same individual.

---

[1] Dujardin-Beaumetz, "De la myélite aiguë," Paris, 1872, p. 121.

[2] In a case of locomotor ataxia under my own care, to be cited more particularly farther on, this retardation amounted to several minutes.

It is not often the case that bed-sores, or other forms of ulceration and death of the soft parts, occur in acute partial myelitis, except in those cases which have a traumatic origin—they are generally rapidly developed. It is not uncommon, however, to witness atrophy of the paralyzed muscles, more or less extensive and complete, according to the extent and profundity of the lesion of the cord.

The temperature of the parts supplied by the nerves coming from the affected region of the cord is, perhaps, in the first place, somewhat increased. Eventually, however, as the paralysis of sensibility and motion becomes more strongly marked, there is a decided fall. By means of Dr. Lombard's thermo-electric differential calorimeter, comparative results can be simultaneously obtained with great ease and exactness.

The electric excitability of the paralyzed parts is always lessened.

The symptoms of acute partial myelitis are, of course, different, according as the cervical, dorsal, or lumbar region, is the seat of the morbid action. For convenience of description, the cord may be divided into two parts: the cervico-dorsal, embracing the cervical region and the dorsal as far as the sixth dorsal vertebra; and the lumbo-dorsal, comprehending the remaining part of the cord.

When the lesion involves the cervico-dorsal region, the upper extremities may be paralyzed without the lower participating; but disturbances of sensibility are generally experienced in all parts situated below the seat of disease. If the upper part of this division be affected there are difficulty of swallowing, disturbances of the respiration and circulation, and gastric derangement. The pupils are at first dilated, and subsequently contracted. Epileptic convulsions are occasionally an accompaniment. There may be, as I have seen in two cases, accessions of great venereal excitement.

When the dorso-lumbar region is the part affected, the lower extremities alone exhibit aberrations of sensibility and motility. The sense of constriction is generally felt, and the bladder and rectum are usually paralyzed.

Acute partial myelitis is much slower in its progress than the general form of the disease, and life may be prolonged for a considerable period if the lesion be not very extensive.

Causes.—Acute myelitis, whether of the general or partial form, is more frequently the result of injury than of any other cause. It is likewise a sequence of disease of the vertebræ, extending to the dura mater and other membranes, and of meningitis. It is also said to be produced by exposure to extreme heat or cold, by violent muscular efforts, and by venereal excesses. Twelve cases have come under my observation. Of these, three were the result of wounds, two ensued on disease of the vertebræ, three on exposure to intense cold, two were apparently due to excessive muscular exertion, and two were caused by extension of acute meningitis.

Diagnosis.—The principal diagnostic marks of acute myelitis are the occurrence of the sensation of constriction around the body, the alkalinity of the urine, the rapid supervention and the completeness of the paralysis, the great predisposition to sloughs wherever there is the least pressure, the excitation of pain in one part of the body by irritation applied to some other part, the causation of reflex movements in a similar way, the speedy loss of electric contractility, and the marked depression of temperature in the paralyzed parts.

From acute meningitis it is distinguished by the fact that the pain in this disease is more severe, that it is aggravated by movements of the spine, and that there are marked, and sometimes permanent, contractions of the limbs. The paralysis is never so profound. Moreover, bed-sores and atrophies are not phenomena met with, except as the results of long-continued pressure in the one case and of disuse in the other.

In congestion of the cord the symptoms are less strongly pronounced, and are more or less subject to remissions; bed-sores are uncommon; the progress of the disease is slower, and the symptoms are aggravated when the patient assumes the recumbent posture, and the urine is not alkaline, except as the consequence of paralysis of the bladder.

From hæmorrhage of the cord the diagnosis is not, in general, a matter of doubt, but the following case, reported by Dujardin-Beaumetz, would seem to present an exception to this statement:

"A porter, while at his work, was suddenly seized with complete paralysis of motion and sensibility of all parts of his body, except the head and neck. There was no loss of consciousness. The bladder and rectum were paralyzed; there were no contractions; the respiration was slow and painful, the diaphragm alone, of all the respiratory muscles, being active. The intelligence was perfect. The diagnosis was hæmorrhage of the cord at about the junction of the cervical with the dorsal portion. Three days after the accession the patient died asphyxiated. On post-mortem examination, no trace of hæmorrhage could be found, but the cord was softened and completely broken up at the dorsal enlargement."

The diagnosis from hysteria may sometimes require to be made

As is well known, this condition may simulate almost every affection of the nervous system, and acute inflammation of the cord is not one of the exceptions. The pain in the back, the constriction around the body, the paraplegia, the cystic and rectal derangements, the anæsthesia as met with in acute myelitis, may all be due to hysteria. But careful examination will serve to make the discrimination easy and complete. The symptoms are exaggerated, and are not constant, the general disturbance of the system is slight, there is no progressive advance of the disease, and the patient, nearly always of the female sex, exhibits the history and diathesis of hysteria so unmistakably, that error is rendered almost out of the question.

Prognosis.—The termination of general acute myelitis is in death sooner or later. Even if it passes into the chronic stage, the alterations in the structure of the cord are so extensive as to be incompatible with the performance of its functions. Death was the result in all the cases that I have personally observed, and this event occurred in all within three weeks.

In partial acute myelitis recovery is not impossible, although even in this form the prognosis is grave, and the life of the patient, if saved, is always at the expense of the sensibility and motility of the parts below the seat of the lesion.

Even then, in many cases, disease of the bladder, and other secondary affections shorten the term of existence.

Morbid Anatomy and Pathology.—In acute general myelitis the whole cord is involved in the morbid action, and exhibits a more or less considerable enlargement throughout its entire length. As both the white and gray substances are implicated, both become broken down by softening, and hence it is impossible to distinguish one from the other. It appears, however, to be extremely probable that originally the morbid process is parenchymatous, that is, confined to the true cell-elements of the cord, and that the neuroglia is subsequently attacked. Extravasations of blood are met with throughout the medullary tissue. The membranes are sometimes adherent to the cord at various points, or there may be puriform accumulations between them and the cord. At other times the pus is found in isolated depots or in canals extending through the entire length of the nerve-substance. The tendency is to a still more decided condition of softening, and eventually a stage is reached in which the cord is reduced to a semi-liquid state.

The inflammation in cases of acute partial myelitis may be limited to the white substance or to the gray substance, or may attack both these tissues. It may likewise affect the antero-lateral columns, the posterior, or extend to both. Undoubtedly, if we had sufficient opportunities to witness cases of spontaneous origin not the result of traumatic causes, or of the extension of other diseases, we should be enabled to distinguish by the symptoms which part of the cord histologically or more frequently the result of injury than of any other cause. It is likewise a sequence of disease of the vertebræ, extending to the dura mater and other membranes, and of meningitis. It is also said to be produced by exposure to extreme heat or cold, by violent muscular efforts, and by venereal excesses. Twelve cases have come under my observation. Of these, three were the result of wounds, two ensued on disease of the vertebræ, three on exposure to intense cold, two were apparently due to excessive muscular exertion, and two were caused by extension of acute meningitis.

Diagnosis.—The principal diagnostic marks of acute myelitis are the occurrence of the sensation of constriction around the body, the alkalinity of the urine, the rapid supervention and the completeness of the paralysis, the great predisposition to sloughs wherever there is the least pressure, the excitation of pain in one part of the body by irritation applied to some other part, the causation of reflex movements in a similar way, the speedy loss of electric contractility, and the marked depression of temperature in the paralyzed parts.

From acute meningitis it is distinguished by the fact that the pain in this disease is more severe, that it is aggravated by movements of the spine, and that there are marked, and sometimes permanent, contractions of the limbs. The paralysis is never so profound. Moreover, bed-sores and atrophies are not phenomena met with, except as the results of long-continued pressure in the one case and of disuse in the other.

In congestion of the cord the symptoms are less strongly pronounced, and are more or less subject to remissions ; bed-sores are uncommon ; the progress of the disease is slower, and the symptoms are aggravated when the patient assumes the recumbent posture, and the urine is not alkaline, except as the consequence of paralysis of the bladder.

From hæmorrhage of the cord the diagnosis is not, in general, a matter of doubt, but the following case, reported by Dujardin-Beaumetz, would seem to present an exception to this statement :

"A porter, while at his work, was suddenly seized with complete paralysis of motion and sensibility of all parts of his body, except the head and neck. There was no loss of consciousness. The bladder and rectum were paralyzed ; there were no contractions ; the respiration was slow and painful, the diaphragm alone, of all the respiratory muscles, being active. The intelligence was perfect. The diagnosis was hæmorrhage of the cord at about the junction of the cervical with the dorsal portion. Three days after the accession the patient died asphyxiated. On post-mortem examination, no trace of hæmorrhage could be found, but the cord was softened and completely broken up at the dorsal enlargement."

The diagnosis from hysteria may sometimes require to be made ment generally does not differ from that recommended in acute meningitis, the indications being almost identical. So far as my experience extends I have never found any means sufficient for cure, and the few successful instances that have been reported are doubtless, as Jaccoud suggests, cases of congestion or meningitis.

But in the partial form of the disease there is some hope of being able to arrest the morbid process, or at least to prevent its extension to the sound parts of the cord. Some authors have recommended mercurials, but I do not perceive any indication for their use. I am satisfied, however, that I have derived decided benefit from the administration of ergot in large doses, as recommended for congestion, and from the employment of revulsives. Of these latter agents the actual cautery occupies the first place. It should be applied either in the form of longitudinal lines on each side of the vertebral column at the seat of the lesion, or as points, to the number of three or four, similarly situated. The skin should be rendered anæsthetic by the ether-spray before the application of the heated metal, and this latter should be platinum, brought to a white heat.

By this agent, in conjunction with the ergot, I have recently, in the case of a carpenter presenting all the symptoms of acute partial myelitis involving the lower dorsal region of the cord, succeeded in effecting such a mitigation of the disease as to arrest its onward progress, and restore motion and sensibility to the paralyzed limbs to quite an appreciable extent. The ergot was administered in doses of a drachm every two hours for five days. Two cauterizations were made during this period. During the ensuing thirty days the ergot was given in similar doses three times daily, and two additional cauterizations were performed. The patient was then left without further medical interference, being able to move his legs, to pass and retain his urine, and to feel impressions made on the skin below the seat of the disease. While complete recovery will not probably result, I am quite satisfied that life was saved by the action of the agents in question.

II.

INFLAMMATION LIMITED TO THE ANTERIOR TRACT OF GRAY MATTER OF THE SPINAL CORD.

I have preferred to include the diseases next to be considered, under the title above given, in preference to others which have been brought forward. Thus the term "*anterior horns of gray matter*" would not apply to the medulla oblongata, and that of "*motor tract*," employed by Dr. E. S. Seguin,[1] does not accord with the views I entertain relative to the physiological anatomy of the region referred to, it being, in my opinion, trophic as well as motor in function. The

[1] "Spinal Paralysis of the Adult," etc., New York, 1874.

topographically is affected. For there can be no doubt that, [illegible]s anæmia, or as we shall see hereafter in certain acute and chronic form of myelitis, the symptoms must be as characteristic as are the function of the several histological and regional parts of the cord.

As regards the obvious morbid anatomical features, we find that when the lesion is situated in the white substance the membranes of the affected portion are congested, thickened, opaque in patches, and adherent to the cord. The cord is softened to a variable depth, and this portion is detached with the membranes if these be removed. This softened portion is in the early stage rose-colored and studded with red points, marking the situation of the enlarged blood-vessels. As the disease advances, the color deepens to a reddish-brown, then begins to get lighter, and, passing through several shades of yellow, eventually becomes white.

When the gray substance is involved, the changes in its physical appearance are similar; and, when both the white and the gray are the seat of the morbid process, it is impossible to distinguish the two substances from each other.

Microscopical examination shows the existence of congestion, and, as an essential feature, an increase in the amount of connective tissue or neuroglia of the cord. The evidences of this hypertrophy are seen in the increase of fusiform cells and in the production of multinuclear cells and free nuclei. These formations take place at the expense of the proper nervous tissue of the cord, the anatomical elements of which undergo atrophy and fatty degeneration. The nervous tubules are thus often disintegrated and their contents disseminated through the extraneous tissue. The axis cylinders are entirely surrounded by oil-globules, or are altogether broken up and rendered unrecognizable.

Should suppuration occur, the elements of pus are observed among those already described, and take their place to a considerable extent.

In case of the passage of acute myelitis into the chronic form, the centre of inflammation usually undergoes other changes, which, however, still maintain the general characteristic of hypertrophy of the neuroglia at the expense of the proper nervous tissue. Induration, or, as it is now generally called, sclerosis, is the result. Occasionally, however, the softening persists and becomes the permanent structural condition of the diseased portion of the cord.

When the lesion is in the gray substance, the microscope shows the nervous cells to be broken up, and the anatomical elements of the blood to be scattered through the tissue.

Treatment.—The treatment of acute general myelitis offers no encouraging features. The most that can be done is to endeavor to prevent, as far as possible, the formation of sloughs, by placing the patient on a water-bed, and by sponging the parts exposed to pressure, with whiskey or with hot and cold water alternately applied. The treatment generally does not differ from that recommended in acute meningitis, the indications being almost identical. So far as my experience extends I have never found any means sufficient for cure, and the few successful instances that have been reported are doubtless, as Jaccoud suggests, cases of congestion or meningitis.

But in the partial form of the disease there is some hope of being able to arrest the morbid process, or at least to prevent its extension to the sound parts of the cord. Some authors have recommended mercurials, but I do not perceive any indication for their use. I am satisfied, however, that I have derived decided benefit from the administration of ergot in large doses, as recommended for congestion, and from the employment of revulsives. Of these latter agents the actual cautery occupies the first place. It should be applied either in the form of longitudinal lines on each side of the vertebral column at the seat of the lesion, or as points, to the number of three or four, similarly situated. The skin should be rendered anæsthetic by the ether-spray before the application of the heated metal, and this latter should be platinum, brought to a white heat.

By this agent, in conjunction with the ergot, I have recently, in the case of a carpenter presenting all the symptoms of acute partial myelitis involving the lower dorsal region of the cord, succeeded in effecting such a mitigation of the disease as to arrest its onward progress, and restore motion and sensibility to the paralyzed limbs to quite an appreciable extent. The ergot was administered in doses of a drachm every two hours for five days. Two cauterizations were made during this period. During the ensuing thirty days the ergot was given in similar doses three times daily, and two additional cauterizations were performed. The patient was then left without further medical interference, being able to move his legs, to pass and retain his urine, and to feel impressions made on the skin below the seat of the disease. While complete recovery will not probably result, I am quite satisfied that life was saved by the action of the agents in question.

II.

INFLAMMATION LIMITED TO THE ANTERIOR TRACT OF GRAY MATTER OF THE SPINAL CORD.

I have preferred to include the diseases next to be considered, under the title above given, in preference to others which have been brought forward. Thus the term "*anterior horns of gray matter*" would not apply to the medulla oblongata, and that of "*motor tract*," employed by Dr. E. S. Seguin,[1] does not accord with the views I entertain relative to the physiological anatomy of the region referred to, it being, in my opinion, trophic as well as motor in function. The term "*anterior tract of gray matter*" is not only sufficiently precise regards the spinal cord proper, but it can logically be applied to th corresponding mass of ganglionic tissue in the medulla oblongata, an at the same time does not commit us in advance to any views relative to the office of this gray matter as a nerve-centre.

---

[1] "Spinal Paralysis of the Adult," etc., New York, 1874.

In inflammation limited to the anterior tract of gray matter of the spinal cord, including the medulla oblongata, the morbid process may involve both the motor and trophic cells—that is, all the nervous elements of which the tissue is composed—or it may be restricted on the one part to the motor cells, and on the other to the trophic cells.

There are, thus, three categories of diseases to be considered under the general head of Inflammation limited to the Anterior Tract of Gray Matter of the Spinal Cord, viz.:

1. Inflammation of motor and trophic nerve-cells :
    a. Infantile spinal paralysis.
    b. Spinal paralysis of adults.
    c. Pseudo-hypertrophic spinal paralysis.

2. Inflammation of the motor-cells:
    a. Glosso-labio-laryngeal paralysis.

3. Inflammation of the trophic cells:
    a. Progressive muscular atrophy.
    b. Progressive facial atrophy.

In addition to these primary affections, there are others in which the anterior tract of gray matter is involved secondarily, or at least in conjunction with inflammation of the white substance, entering into the composition of the antero-lateral columns of the cord. These will be considered under another head.

1. *Inflammation of Motor and Trophic Nerve-Cells.*

All the diseases of this class are characterized by two essential phenomena, paralysis and atrophy. The paralysis is the first of these symptoms to make its appearance, the atrophy following more or less closely, and ensuing not as a consequence of paralysis and disuse, but as an active pathological condition. The chief reasons, as we shall see hereafter, for the theory of the existence of trophic cells in the spinal cord, are found in the facts that the atrophy is an independent feature of the diseases of the class under notice, and that it may exist without paralysis at all, except in so far as an atrophied muscle is necessarily weaker than one not so affected; and, again, that paralysis may exist without atrophy, and the gray matter of the anterior tract alone be involved.

a. Infantile Spinal Paralysis—Organic Infantile Paralysis.

Under the name of organic infantile paralysis—to which term, now that the morbid anatomy is well understood, that of infantile spinal paralysis is to be preferred—I have considered at length[1] a form of paralysis occurring in young children, previously described by Heine,[2] who was the first to direct special attention to it under the name used at the head of this section; by Rilliet[3] and Barthez as the *paralysie essentielle de l'enfance*, and by Duchenne[4] as *paralysie atrophique graisseuse de l'enfance.* Previous to the writings of these authors, the affection in question was not distinctly recognized as a separate disease, but was confounded with a much less serious disorder, probably belonging to the class already considered under the head of anæmia of the anterior columns of the spinal cord. The tendency in the present affection to muscular atrophy, and the permanent character of the paralysis, are phenomena which sufficiently distinguish it from the temporary paralysis referred to.

Symptoms.—The beginning of infantile spinal paralysis is generally indicated by febrile excitement, convulsions, and pain in the back. This pain marks the seat of the disease in the spinal cord to which the paralysis of the muscles is due. These symptoms last for a few days, or they may be so slight as in very young children not to attract attention; or, again, they may be absent altogether.

Sometimes the paralysis is readily observed from the first, both by its extent and intensity; at others, it is not perceived till some one notices that the child does not use one hand or kick with one leg. The age of the patient, of course, exercises considerable influence on the question of ascertaining the existence of the paralysis at an early period. All four of the limbs may be affected, or the paralysis may be restricted to the legs, or more rarely to the arms, or to one arm and one leg of the same side, or of opposite sides, or to one leg or one arm, or even to a group of muscles or a single muscle.

The temperature of the affected limbs is always much lower than that of the corresponding sound ones. The difference is sometimes as much as eight or ten degrees, though generally it is not more than five. If, spontaneously or under appropriate treatment, amendment takes place, the first indication is shown by the return of the temperature toward the natural standard. It thus becomes important to have some means by which a very slight increase of heat may be noticed. A delicate thermometer graduated to tenths of a degree will generally suffice, but much more exact indications may be obtained by Lombard's thermo-electric differential calorimeter, described in the introduction to this treatise. One of the thermo-electric piles is placed on the sound limb, the other on the corresponding part of the paralyzed limb. Both are in connection, by delicate silk-covered wire, with the poles of a galvanometer. If the temperature of both limbs be the same, the needle of the galvanometer remains quiet. If either be warmer than the other, the needle is deflected to the north or the south, according as one or the other limb has the higher temperature. By this apparatus, very small fractions of a degree of temperature can be determined with absolute certainty.

---

[1] *Journal of Psychological Medicine*, No. 1, 1867, p. 49. Also, my translation of Meyer's "Electricity in its Relations to Practical Medicine," New York, 1870, p. 228, note.

[2] "Beobachtungen über Lähmungszustände der untern Extremitäten und deren Behandlung," Stuttgart, 1840; and "Spinale Kinderlähmung," 2d Aufl., Stuttgart, 1860.

[3] "Traité, clinique et pratique, des maladies de l'enfance," Paris, 1853, tome ii., p. 335.

[4] *Gazette hebdomadaire*, 1845, and "Traité de l'électrisation localisée," 1re édition, Paris, 1855.

Sensibility is not materially, if at all, lessened, though the reflex excitability is diminished, and often entirely abolished, from the very first.

The faradaic current almost always fails from the earliest period to cause contractions in the paralyzed muscles, but the galvanic current will, even when of low tension, produce movements in the most thoroughly paralyzed muscles, before the stage of atrophy is reached. This first period of infantile spinal paralysis, in which the loss of power is the most obvious symptom, may last a month, or even six months, before the second period, characterized by atrophy, begins. It is then usually the case that the paralysis gradually disappears to a great extent, if the loss of motor power has in the first place been extensive. Even when the paralysis has been restricted to a single limb, some muscles regain their function, and in either case complete restoration may occur. In those parts, however, in which there is no retrogression of the disease, atrophy ensues, and advances sometimes with great rapidity. The temperature falls still lower, till, in some cases, it is scarcely higher than that of the surrounding atmosphere. In a patient from Maine, a little girl of about ten years of age, in whom both the lower extremities remained paralyzed, and were atrophied to a very marked degree, the temperature of the legs below the knee was only 75° Fahr. in an atmosphere of 72°. The skin is of a livid hue, and pressure with the point of the finger causes a white spot to appear, which does not again become colored for some time, owing to the torpidity of the capillary circulation.

With this atrophy, the electric contractility of the muscles disappears, although it has begun to be lost at an earlier period, and hence the strongest induced currents fail to cause the slightest contraction, and in some cases even powerful primary currents are equally inefficacious. Indeed, in no other disease is the electric excitability so thoroughly abolished as in that under consideration.

Owing to the atrophy and consequent weakness of the muscles which surround the articulations, as well as to relaxation of the ligaments of the paralyzed limbs, the bones entering into the composition of the joints become separated. This condition is especially manifested when the upper extremity is the affected part, as regards the shoulder, the head of the humerus sometimes falling away from the glenoid cavity to the extent of an inch or more. The passive mobility of the joint is therefore very greatly increased, and dislocation is readily effected.

If, as is often the case, certain muscles of a limb regain their power, while others remain paralyzed, the normal equilibrium is destroyed, and distortions of various kinds are consequently produced. Hence, infantile spinal paralysis is among the most important causes of club-feet.

The bones are also subject to atrophy and to arrest of growth, and therefore the paralyzed and atrophied limb eventually is shorter than the corresponding sound member. In the case of a boy, six years old, who, several years since, was under my charge, the left arm, in consequence of infantile spinal paralysis occurring in his second year, was two inches shorter than the right. This arrest of growth was not very evident when the child was dressed, and the limb, by its own weight, hung by the side, for the reason that the head of the humerus was separated nearly two inches from the glenoid cavity, but, when the bones were brought into apposition, the shortening was of course apparent. This extension of the atrophy and arrest of development to the osseous system is by no means an invariable accompaniment, and is perhaps never produced unless the original central lesion is profound, and the muscles, generally, of an extremity are involved.

Unless death should occur during the first stage of the disease, it is not probable that spinal infantile paralysis will in any case tend to shorten life. The tendency is for the spinal lesion to limit itself, and hence, when the second stage of the disease appears, there is no probability that any extension of the morbid process will take place. The consequences are entirely restricted to the parts which are in nervous relation with the region of the cord in which the central lesion exists.

At no time during the course of spinal infantile paralysis is either the bladder or its sphincter paralyzed, neither is the sphincter ani deprived of its contractile power.

The muscles most apt, according to my experience, to become the ultimate seat of the paralysis and atrophy are the tibialis anticus, the peroneal, the deltoid, the gluteal, the extensors of the toes, and the quadriceps femoris. I have never seen a case in which any muscle of the head or neck was involved. Seguin[1] states that the temporal has been found paralyzed once. Bed-sores or atrophic ulcerations of the skin rarely occur. I have never observed a case in which they were present —a fact which goes to show that, notwithstanding the appearance of the surface over the paralyzed parts, the nutrition of the skin is not essentially lessened.

---

[1] "Infantile Spinal Paralysis," *Medical Record*, January 15, 1874.

Causes.—Little is known of the etiology of infantile spinal paralysis. In two cases under my observation, occurring in brothers, it was apparently induced by the nurse allowing the infants to lie on the damp ground for an hour or more; in several other cases, it came on while the children were suffering from teething, and in others it has followed diseases of various kinds, such as whooping-cough, measles, scarlet fever, etc. In the great majority of the cases that I have witnessed, no cause could be reasonably assigned.

More than half of the cases occur during the first two years of life. M. Duchenne (de Boulogne), the younger,[1] of fifty-six cases occurring in the private practice of his father, finds the proportion of cases, for the several ages up to ten years, as follows:

Twelve days after birth	1
At the age of one month	1
At two months	2
At from four to six months	6
At from six months to a year	6
From one year to eighteen months	20
From eighteen months to two years	11
From two to three years	5
From three to four years	2
At seven years	1
At ten years	1
Total	56

Diagnosis.—The symptoms of infantile spinal paralysis in the early part of its first stage are rarely so characteristic as to admit of a rational diagnosis being given. They are such as are met with in many other affections, and the early age of the patient is usually an obstacle to exact inquiries. I shall, under the head of morbid anatomy, cite cases in which spinal hæmorrhage has produced symptoms in some respects similar to those of infantile spinal paralysis, but such cases are extremely rare, and they are not characterized by the progressive atrophy and marked reduction of temperature so characteristic of the affection under notice. Setting them aside, it is not probable that, having in view the phenomena of the disease, the intelligent physician of the present day will blunder in his diagnosis. The absence of cerebral symptoms, the cessation of the fever when it has existed, and the general good health of the patient, will go to render the diagnosis still more certain. The only condition with which the disease in question may be confounded, is the temporary paralysis due to reflex irritations, and probably the direct consequence of spinal anæmia. But the fact that such irritations are generally sufficiently evident, and that the paralysis disappears with their removal, will not permit us to remain long in doubt. As the disease advances to its full development, the symptoms become more and more characteristic, until doubt is scarcely any longer possible. In fact, in its entirety, infantile spinal paralysis cannot be mistaken for any other affection.

---

[1] Duchenne (de Boulogne), "De l'electrisation localisée," troisième édition, Paris, 1872, p. 417.

Prognosis.—Infantile spinal paralysis is not an affection liable to terminate fatally. Death may possibly occur in the very inception of the disorder from the irritation and general disturbance due to the inflammation of the cord, but, though I admit the possibility of such an event, none such has ever come under my observation, nor have I been able to find any such recorded. The prognosis is therefore only of importance as regards the consequent paralysis and atrophy. And here it depends very much upon the fact as to whether the disease has advanced so far as to have resulted in the abolition of the electric contractility of the affected muscles. If this is lost to the induced current, the cure will be difficult, and the treatment protracted; if the primary current is also powerless, a cure is impossible. I believe I was the first to use the primary current in the treatment of infantile paralysis, and to insist on its great value as a curative agent, and as an element in the prognosis.[1] If the muscles can be made to contract with either the induced or primary currents, the cure is often merely a matter of time and patience. But regard must also be had to the extent of the paralysis and atrophy. If all the muscles of one or more of the limbs are involved, and if contractions in the non-affected muscles have interfered to any considerable extent with the conformation of the joints, a cure will be next to impossible. While, therefore, recognizing the severity of the lesions in infantile spinal paralysis, and the tediousness of the methods of cure, I cannot look upon the affection with the hopelessness of Volkmann.[2] For with Dr. Radcliffe[3] I am every day more and more convinced that muscles which I should once have looked upon as hopelessly paralyzed, may be resuscitated by proper treatment.

Again, it must not be forgotten that the most extensive paralysis, in the disease under consideration, may in great part, or entirely, spontaneously disappear before the atrophy begins to make its appearance. It is not, therefore, safe to venture on a prediction as to the ultimate result at any time anterior to the stage of atrophy.

Morbid Anatomy.—The morbid anatomy of infantile spinal paralysis is to be studied in the spinal cord, the nerves, the muscles, and the bones—the lesions in the three latter tissues being secondary to those existing in the cord. Previous to the recent investigations of Vulpian and Prévost, Dr. Lockhart Clarke, and Charcot and his pupils, there was no approach to uniformity relative to the essential character of the disease, many observers denying that there was any structural central lesion. Even since this last-named distinguished observer, in conjunction with Joffroy, published the report of his notable case, with a detailed statement of the post-mortem appearances, and since his results have been confirmed by others, we find so prominent a teacher and physician as Dr. West[1] ignoring them altogether, and concentrating his attention entirely on the eccentric lesions in a few brief sentences.

---

[1] *New York Medical Journal*, December, 1865.

[2] "Ueber Kinderlähmung und paralytische Contracturen-Sammlung," Klinische Vorträge, No. 1, Leipzig, 1870.

[3] Reynolds's "System of Medicine," vol. iii., p. 666.

It is not to be denied that paralysis of spinal origin may exist in children and be a very different affection from the one under notice. Paralysis, like cough, is only a symptom which may be due to many very different lesions. Thus, in a case of paralysis in a child six years of age, which had begun four years previously, and which involved the left lower extremity, I had the opportunity of making a post-mortem examination—death occurring from pneumonia. On examining the spinal cord, I found in the lower part of the dorsal region, and in the left anterior column, a cicatrix partially filled with a very small clot. No microscopical examination was made, and hence the condition of the anterior cornua was not ascertained. The atrophy of the paralyzed muscles was very slight, and it is therefore possible that there was no primary lesion of the nerve-cells of the anterior horns. The paralysis had ensued suddenly, and may have followed a fall or a blow—no accurate history could be obtained. I then, and for some time subsequently, regarded this case as one of infantile spinal paralysis as at present understood, but I am now entirely satisfied that, beyond the loss of motor power, it had little in common with this affection. The slight atrophy which existed was possibly the result of secondary degeneration of a few cells of the left anterior horn, and not a consequence of any primary lesion of this region. A histological examination would have done much toward the elucidation of this interesting case, but it was at the time impossible.

Dr. Clifford Allbutt[2] has reported a case in which the symptoms were more clearly the result of hæmorrhage. The patient was an infant in good health, seven months old. One evening the mother lifted the child rather suddenly, and was astonished to see the body fall heavily forward. There were no evidences of pain, but she shortly afterward perceived that it was paralyzed in all four limbs. Death ensued in a short time from implication of the respiratory nerves. The spinal cord was submitted to careful examination, and two hæmorrhagic clots were discovered in the cervical region. One of these, of small size, was in the left posterior horn; the other, larger, was in the right posterior horn and lateral column. If these clots had been formed in the lower dorsal region the infant would probably have survived, and the case might have been regarded as one of infantile spinal paralysis.

---

[1] "On some Disorders of the Nervous System in Childhood"—being the Lumleian Lectures for 1871, Philadelphia, 1871, p. 87.

[2] The *Lancet*, vol. ii., 1870, p. 84.

In a case reported by Hayem,[1] the patient was attacked with paralysis of the lower extremities at the age of two years. Death took place twenty-two years afterward, of phthisis. The gray substance of the cord contained blood-pigment disseminated through its substance.

Such instances, as I have said, only go to show the similarity of symptoms which may result from very different causes, and like examples will readily occur to the reader as being afforded by unlike lesions in other parts of the body.

The first attempt to associate spinal infantile paralysis with lesion of the anterior horns of the spinal cord was made by Cornil,[1] who reported the case of a patient affected with the disease in question, who died of cancer of the mammary gland at the age of forty-nine. The affection had been contracted by the subject, when an infant two years old, being left to lie for a long time on cold and damp ground. The muscles of the inferior extremities, especially those of the left, were paralyzed and atrophied. The post-mortem examination, which extended to the muscles, the nerves, and the spinal cord, revealed the existence in this latter organ of atrophy of the anterior horns of gray matter and of the antero-lateral columns—in those parts of the cord from which emanated the nerves going to the affected muscles. This case was the first published, in which lesion of the cord was noted in connection with infantile spinal paralysis, though the author states that he had previously, in 1863, observed an increased development of connective tissue in the anterior columns. The case of hæmorrhage coming under my own notice, previously cited, occurred in 1858.

Prévost[2] described, in 1865, the case of a woman, aged seventy-eight, in whom there was paralysis of the left leg, with deformation of the foot, evidently, in the opinion of M. Vulpian, whose patient she was in the Salpêtrière, the result of infantile spinal paralysis. The muscles of the left leg and foot, as well as those of the lower part of the thigh, were much atrophied. The patient was demented, and died of phthisis. Post-mortem examination showed the left anterior horn of gray matter to be atrophied. On microscopical examination, it was seen that all the external part of this horn had undergone an alteration, the nerve-cells being replaced by a cellular and nuclear tissue evidently the proliferation of the neuroglia. This was colored red by carmine. Amyloid corpuscles were also present. The ganglion-cells of this part had almost entirely disappeared, and the one or two that remained were atrophied. The cells of the internal group were also diminished in number. The right anterior horn was normal.

---

[1] "Comptes rendus des séances, et mémoires de la société de biologie," 1869, 1870.

[2] Ibid., tome v., série iii., 1863, p. 187.

This was the first case in which atrophy and disappearance of the cells of the anterior horn were found associated with infantile spinal paralysis.

In 1868, Dr. Lockhart Clarke,[1] in collaboration with Mr. Z. Johnson, published, under the head of muscular atrophy, the details of a case which was clearly one of infantile spinal paralysis. The disease had ensued in early infancy, immediately after inoculation with small-pox virus, and involved both upper extremities, which, besides being paralyzed, were greatly atrophied. Examination of the cord showed atrophy and softening of both anterior horns, with atrophy and degeneration of nerve-cells. In many places the cells had disappeared.

Then in 1870,[2] Charcot, in conjunction with his pupil Joffroy, gave the results of his examination of a case which may be considered as definitely settling the question of the morbid anatomy of infantile spinal paralysis. The patient, a woman named Wilson, died at the age of forty-five years, of phthisis, having been the subject of paralysis since childhood. The disease had suddenly made its appearance when she was seven years old, and had at first involved all four limbs. At the end of a year the upper extremities had in a measure regained their power, the lower remained atrophied and nearly altogether paralyzed.

On post-mortem examination the spinal cord was found to be affected from the cervical to the lumbar enlargement. The alterations were chiefly in the gray matter, and especially in the anterior cornua. These were atrophied and distorted, and the cells had disappeared to a very great extent. In some places entire groups of cells had disappeared, without leaving any traces of their former presence. In the immediate vicinity of some of the points of cellular atrophy, the neuroglia had undergone sclerous transformation, but there were places where the lesion of the cells was the only alteration which could be discovered.

Since the publication of the details of Charcot's case, several others have been reported, and a number of excellent monographs have been written in illustration of the morbid anatomy of infantile spinal paralysis. Among these may be cited those of Parrot and Joffroy,[3] Roger and Damaschino,[4] Dujardin-Beaumetz,[5] Petitfils,[6] Seguin,[7] Putnam-Jacobi,[8]

---

[1] "On a Remarkable Case of Extreme Muscular Atrophy, with Extensive Disease of the Spinal Cord," "Medico-Chirurgical Transactions," Second Series, vol. xxxiii., 1868, p. 249.

[2] "Archives de Physiologie," tome iii., 1870, p. 135.

[3] Ibid., 1870, p. 310.

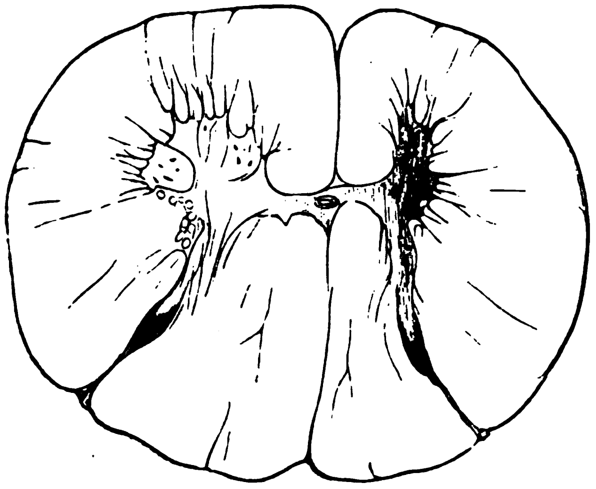
[4] "Recherches anatomo-pathologiques sur la paralysie de l'enfance," *Gazette Médicale de Paris*, 1871, Nos. 41, 43, 45, 48, and 51. [5] "De la myelite aiguë," Paris, 1873.

[6] "Considérations sur l'atrophie des cellules motrices," Paris, 1873.

[7] "Infantile Spinal Paralysis," *Medical Record*, January 15, 1874.

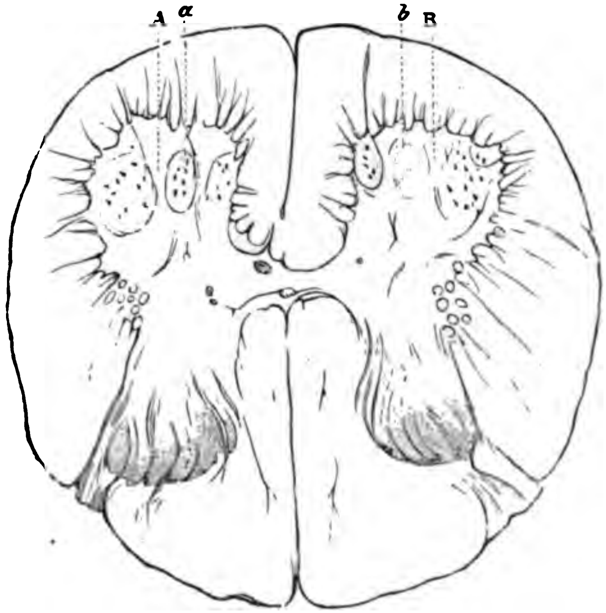
[8] *American Journal of Obstetrics*, May, 1874.

FIG. 88.



harcot,[1] who has quite recently traversed the whole ground, and as admirably summed up what is known of the whole subject.

FIG. 89.



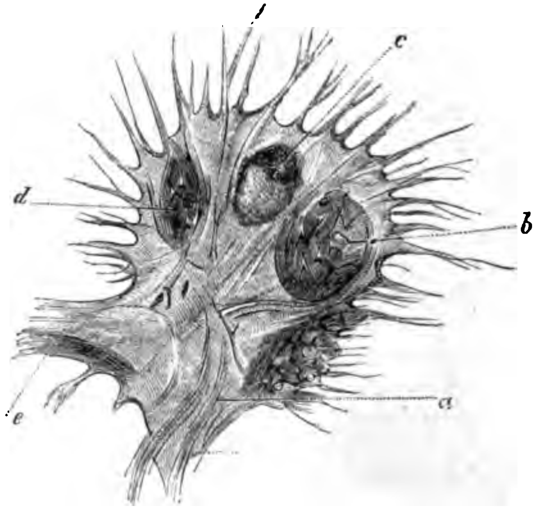
---

ue *photographique des hôpitaux*, Janvier et Février, 1872, and "Leçons sur les du système nerveux," fascicule iii., Paris, 1874.

They all go to show that the essential lesion in infantile spinal paralysis is situated in the anterior horns of gray matter, and that it consists of a myelitis, in consequence of which there is an atrophy of the part affected, a degeneration of its structure, and a disappearance of its cell-elements. This contraction or atrophy is well shown in the accompanying woodcut from Charcot (Fig. 38), which represents a magnified section of the spinal cord taken from the cervical region of a woman, aged fifty years, who died in the Salpêtrière, of general paralysis of the insane, and in whom there was infantile spinal paralysis affecting the right superior extremity. The atrophy of the right anterior horn is well marked, and the emaciation of the right antero-lateral and posterior columns, probably a secondary complication, is also notable.

The atrophy and disappearance of the nerve-cells are sometimes exceedingly limited. In the accompanying figure (Fig. 39), also from Charcot, an enlarged view is given of a section of the spinal cord taken from the lumbar region in a case of infantile spinal paralysis, affecting the right lower extremity : *A*, the left anterior horn, healthy ; *a*, healthy group of ganglion-cells ; *B*, right anterior horn ; *b*, median ganglionary nucleus, of which the cells are destroyed, and which is represented by a *foyer* of sclerosis. In Fig. 40, a still more enlarged view is given of the right anterior horn : *a*, cervix of the posterior horn ; *b*, postero-external group of nerve-cells ; *c*, antero-external group, the cells of which have entirely disappeared, while they are intact in groups *b* and *d* ; *d*, internal group ; *e*, the commissure.

Fig. 40.



The myelitis is parenchymatous in character, that is, it begins in the nerve-cell structure, and, if the neuroglia be found involved, it is from the extension of the morbid process, and not from any primary implication. This is sufficiently established, not only from an examination of sections of the cord, such as that represented in the last figure in which the lesion is restricted to the nervous elements, but from a consideration of the physiological relation which exists between the cells of the anterior horn and the functions which they have to perform—functions which are interfered with in cases of infantile spinal paralysis.

Roger and Damaschino[1] have had the opportunity of making histological examinations in three cases of infantile spinal paralysis, in which death took place from intercurrent affections while the disease was still in its early stage. As the result of their observations they conclude that—

"1. The characteristic alteration of infantile paralysis is a lesion of the spinal cord, of which the atrophy of the nerves and muscles is the consequence.

"2. This lesion is more particularly seated in the anterior portion of the gray spinal substance, where it is seen in the form of centres of softening.

"3. This softening is of an inflammatory character, and the disease is a myelitis.

"4. Infantile paralysis ought therefore to be called *infantile spinal paralysis*, and moreover its nosological position is certainly among the affections of the cord, and among the myelites."

As regards the cell-alterations they found them to consist in atrophy, with pigmentation.

Charcot[2] has figured the changes which the cells of the anterior horns undergo in such cases: *A* represents the normal state; *B*, a cell hypertrophied; *C*, pigmentary alteration of the last stage of pigmentary change; *E*, a cell in a state of sclerous atrophy; and *F*, vacuolary alteration, which latter may be the result of the processes used in preparing the specimens—Fig. 41.

The anterior roots of the nerves coming from the affected region have been found atrophied, the myeline having disappeared, and only the axis-cylinder remaining. In other cases the nerve-tubules have been found to be very attenuated, and separated from each other by large spaces filled with connective tissue.

The ganglia of the sympathetic have been examined by Roger and Damaschino, but exhibited no change from their normal structure.

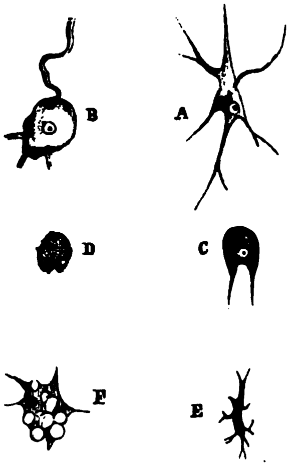
The bones of the paralyzed parts undergo atrophy with the muscles, though, when the lesion is not extensive, the bony atrophy may escape recognition. We have already seen that the affected extremities are often submitted to an arrest or retardation of growth. Besides this condition, there is a cessation in the development of the bone laterally, and consequently its shaft remains smaller than is natural. The articular extremities of the affected bones lose their cartilages, and are more or less arrested in their development. Examined microscopically, as has been done by Laborde[1] and others, the osseous tissue is found to present a deficient number of bone-cells and an excessive amount of medullary elements and adipose matter. It does not appear that the normal relation of earthy to animal matter is disturbed to such an extent as to render the bones either especially liable to fracture or distortion.

---

[1] *Op. cit.*

[2] Leçons sur les maladies du système nerveux, troisième partie, Paris, 1874, p. 184.

FIG. 41.



But, of all the peripheric lesions, those of the muscles have attracted the most attention, and have been the most carefully studied. It appears to be settled without doubt that the first stage of atrophy is characterized by a diminution of the diameter of the muscular fibrillæ, and that there is not then any histological evidence of a tendency to fatty degeneration.

At this time there is an increased formation of connective tissue—a process which appears to persist for a considerable period.

Eventually the atrophied muscles tend, in the great majority of cases, to break down into fat. The transverse striæ disappear, and the degeneration, at first granular and bony, becomes unmistakably fatty. Eventually the muscle consists of nothing but fat and connective tissue, and in time the former disappears, leaving only a mass composed of the sarcolemmæ and connective tissue.

The nature of the morbid process is well shown in the accompanying woodcuts, made from my own drawings of the microscopical appear-of portions of diseased muscles removed by Duchenne's trocar. 42 represents a portion of the upper part of the tibialis anticus e of a boy who had suffered from organic infantile paralysis for two years, and in whom the progress of the atrophy was exceed- rapid. Oil-globules are seen along the course of the fibrillæ. latter are irregular and torn, and the transverse striæ are becom- im.

---

[1] "De la paralysie essentielle de l'enfance," Thèse de Paris, 1864, p. 30.

Fig. 42.

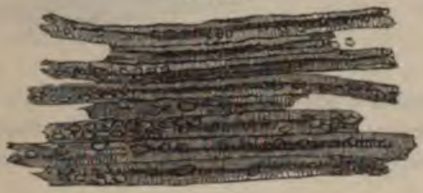


Fig. 43 a still more advanced stage is shown. This cut repre- a portion of the same muscle taken from the lower part. The rerse striæ have nearly disappeared, oil-globules are seen in large ers, and fat-corpuscles are also abundant.

Fig. 43.

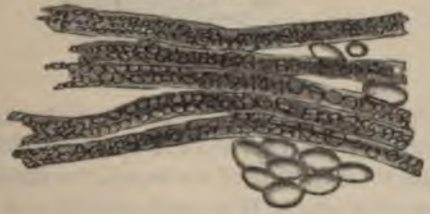


Fig. 44 the progress of the disease is well shown. The upper n of the specimen is a mass of fat-globules, and throughout the the transverse striæ are absent.

Fig. 44.

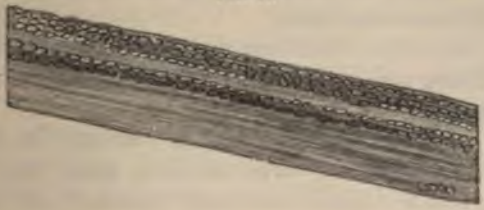


Fig. 45 is shown a portion taken from the same muscle one month the preceding specimens were removed. The transverse striæ are ly gone, and the muscle is a mass of oil-globules and fat-vesi-

FIG. 45.

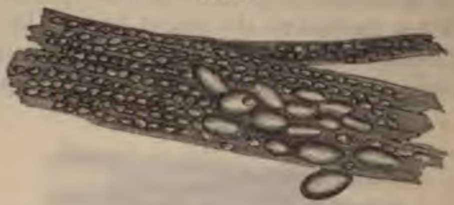
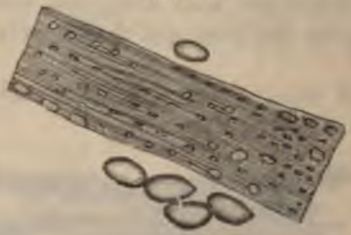


Fig. 46 represents a piece of the same muscle six weeks later. It is now nothing more than a mass of connective tissue, the fat being almost entirely absorbed; no transverse or longitudinal striæ are to be perceived.

FIG. 46.



But there is not, as Duchenne affirms, this degeneration in every case of organic infantile paralysis. In two cases, which had lasted over four years, I found the structure of the muscle unchanged. There were atrophy, loss of electric contractility, and reduction of temperature, but every specimen of the affected muscles that I examined showed no change from the normal character. In every other respect the symptoms were similar to those observed in ordinary cases of the disease. Improvement was very slow, but finally every muscle except the rectus femoris in one, and the tibialis anticus in the other, recovered, and the children were enabled to walk. The affection in both cases was confined to the left lower extremity.

I am hence led to the conclusion that fatty degeneration of muscles, though the ordinary result of organic infantile paralysis, is not an invariable consequence.[1]

Pathology.—Whether all the cells of the anterior horns of gray matter are motor, or whether there are both motor and trophic cells, are questions which the histological examination of the normal structure seems to be quite incapable of satisfactorily answering. Samuel[1] has contended for the existence of a distinct system of nerves, the function of which is to preside over the nutrition of the parts to which they are distributed, and there is not wanting physiological evidence to support his theory; as, for instance, the troubles of nutrition which result in the eye when the fifth pair is divided, and which Vulpian[2] admits are due neither to irritation of the divided nerve-fibres nor to paralysis of the vaso-motor fibres contained in the nerve. Waller[3] has also expressed his opinion relative to the existence of distinct trophic centres in the cord. He regarded the ganglion of the posterior root as the trophic centre for this root, while the gray substance of the anterior horns is the trophic centre for the anterior root. In regard to this theory, Weir Mitchell[4] expresses the opinion that it is still a matter of doubt, in which view all will unite till actual demonstration settles it affirmatively or negatively.

---

[1] *Journal of Psychological Medicine*, No. 1, 1867, p. 57. Since the observations then published, other observers have arrived at the same conclusion. Thus, M. Charcot (*Op. cit.*, p. 161) says: "The surcharge of fat, although habitual in old cases of infantile muscular atrophy, is nevertheless not necessary. By the side of the muscles distended with fat, there are often others which are reduced to a very small volume, and in which the adipose tissue is almost entirely absent. In these last are found primitive fasciculi of very small diameter, but possessing their characteristic striation."

But pathology points still more clearly than does physiology to the existence of trophic cells in the spinal cord. In infantile spinal paralysis the peripheric disturbance is, in the first place, solely one of motility; there is paralysis without atrophy. After a time, which may be as much as six months, or even more, the trophic changes begin. These, as we have seen, are not of that mild character resulting from disuse, but are active and intense, leading to the certain destruction of whole groups of muscles, and even to arrest of development and degeneration of the bones. It is impossible, it appears to me, to account satisfactorily for this atrophic process on the supposition that all the cells of the anterior horns of gray matter are motor, and that they alone are involved in the lesion. Charcot, however, constantly speaks of the affection in question as essentially consisting in an atrophy and disappearance of motor nerve-cells, and the majority of French writers adopt his view. Indeed, he argues very strenuously against the existence of spinal trophic cells, in which, it appears to me, he ignores some of the most valuable contributions which he and others of his countrymen have made to the pathology of the nervous system. A very important memoir of MM. Duchenne and Joffroy[5] throws much light upon this interesting subject, and will be fully considered under the head of progressive muscular atrophy, when additional evidence in support of the theory of the existence of trophic cells will be adduced.

But, whether we admit the existence of trophic cells in the anterior horns of gray matter or not, there is no doubt of the dependence of the

[1] "Die tropischen Nerven," Leipzig, 1860.

[2] "Leçons sur l'appareil vaso-moteur," Paris, 1875, tome ii., p. 377.

[3] "Proceedings of the Royal Society of London," vol. ii., 1860–'62.

[4] "Injuries of Nerves and their Consequences," Philadelphia, 1872, p. 78.

[5] "De l'atrophie aiguë et chronique des cellules nerveuses," etc., *Archives de physiologie*, No. 4, 1870, p. 499.

peripheric troubles on the central lesion. Some authors have assumed that the essential feature of the disease was some disturbance in the sympathetic nervous system; but there is no evidence to support this view. On the contrary, examination has shown that there is no appreciable lesion of this system, and the fact that all the functions of the organism are generally well performed in cases of infantile spinal paralysis militates strongly against the hypothesis.

No examination of the cord of a patient dying during the very earliest stage of infantile spinal paralysis has yet been made. Judging, however, from the character of the symptoms, and from their diffusion, and subsequent retrogression, it is extremely probable that, as in other inflammatory affections, there is congestion, and that this condition is not limited to the anterior tract of gray matter. As we have seen, pains not only in the cord, but in the limbs, are occasionally met with, and Vulpian[1] refers to an instance in which there was complete anæsthesia. In the case of a little girl whom he examined a few days after the invasion of the disease, and in whom the electro-muscular contractility of the muscles of both inferior extremities was entirely abolished to strong faradaic currents, sensibility was equally annihilated, so that the electric brush could be passed over the skin without pain being produced.

The pathology of the deformations so generally met with in cases of infantile spinal paralysis is very obviously the result of the destruction or impairment of that normal equilibrium which exists between the muscles. Thus, if the extensors of the hand are affected while the flexors remain unparalyzed, these latter will in time cause a flexion of the hand upon the forearm ; if the muscles of one side only of the spine are paralyzed, the muscles of the other side will produce a lateral curvature; if the extensors of the foot are alone deprived of their power, the strong gastrocnemius and soleus cause a talipes equinus; while, if these latter are the seat of the derangement while the extensors are healthy, a talipes calcaneus is the result ; and these conditions are more or less modified according as other muscles are more or less involved.

Treatment.—The fact that infantile spinal paralysis is due to an organic affection of the spinal cord is no bar to treatment addressed to the peripheric lesions—it having been very definitely shown by numerous investigations that the integrity of nerve-centres is affected either favorably or unfavorably by eccentric nerve-conditions. It is therefore perfectly practicable, in favorable cases of the disease in question, so to improve the nutrition of the cord, by proper measures directed to the relief of the peripheric trouble, as to arrest the morbid process in the cells of the anterior horns, and even to effect their regeneration. The fact that cases of long-standing infantile spinal paralysis are cured—cases in which there can be no doubt of the existence of the spinal lesion—is of itself sufficient evidence to establish the correctness of the view advanced. The investigations of MM. Masius and Van Lair,[1] relative to the regeneration of the spinal cord, also show how great is the reparative power of the organ. They divided the cord in frogs, and at the end of from two to four months obtained indubitable evidence that the animals had regained voluntary movements and sensibility in the posterior extremities. In other frogs, histological examination showed the more or less complete regeneration of the cord. The conditions which lead us to expect a favorable or an unfavorable result from treatment are stated under the head of prognosis.

---

[1] "Leçons sur l'appareil vaso-moteur," Paris, 1875, tome ii., p. 410.

The treatment of the disease, however, consists both in the use of general and local means. Of the former, ergot is chief, and should be given as soon as we can determine the nature of the disease under which the child is suffering. Young children bear this remedy well. Infants of six months may take as much as ten drops of the fluid-extract three times a day, and this may be increased to half a drachm for children of from one to two years. It is rarely the case, however, that we have the opportunity of giving this valuable agent from the very inception of the disease. But even after the first or febrile stage has subsided, when the affection is solely manifested by paralysis, before the atrophic stage has begun, ergot is of great service—not to be surpassed, in my opinion, by any other medicine, and the only one capable of cutting short the disease, or lessening its extent.

After the stage of atrophy is reached there is no longer any benefit to be derived from ergot; strychnia is then useful because it is capable of acting as a general stimulant to the nervous system, is possessed of undoubted value in cases of degeneration of nervous tissue, and is, moreover, a tonic to the muscles. I generally prescribe it in union with iron and phosphoric acid, according to the following formula: ℞. Strychniæ sul. gr. j, ferri pyrophosph. ʒ ss., acidi phosphorici ℥ ss., syrupus zingiberis ℥ iijss. M. ft. mist. Dose, a teaspoonful or less, according to the age of the patient. A child of from three to five years of age can take half a teaspoonful of this mixture thrice daily; or, the strychnia may be given advantageously in the form of hypodermic injections in doses suitable to the age. In children under one year old, the ninety-sixth of a grain is as much as should be given at a dose, and under six months it should not be administered at all. I am quite sure that strychnia, hypodermically introduced in very gradually-increased doses, is more efficacious than when taken into the stomach.

The immediately local means of treatment are those which are calculated to promote the nutrition of the muscles, and restore or augment their contractile power. The first end is effected by causing a greater amount of blood to flow through the diseased parts; the second is best accomplished by the persistent use of electricity, and active and passive exercise.

---

[1] "Recherches expérimentales sur la régénération anatomique et fonctionnelle de la moelle épinière," analyzed in *Archives de Physiologie*, tome iv., p. 268.

Under the first head are embraced heat, friction, and kneading.

Heat is best applied by means of hot water. A temperature of from 110° to 120° Fahr. may be used, and the limb should be thoroughly immersed, and allowed to remain so for half an hour; salt may be added to the water, with the view of augmenting the stimulant effect.

Frictions with a dry towel, a flesh-brush, or the hand, are also exceedingly useful; they should be practised several times in the course of the day, to the extent of reddening the skin.

Kneading the muscles affords a means of exercising them, and of increasing the amount of blood in the vessels. They should be pinched firmly between the fingers of both hands to the extent of producing some little pain; every paralyzed muscle should be gone over in this way daily.

Jounod's boot, when the inferior extremity is the one affected, or a similar apparatus for the upper extremity, is an efficacious means of causing an increased flow of blood to the parts, and of producing a permanent enlargement of the vessels. Care, however, should be taken that the exhaustion of the air be not carried too far.

Under the second head, electricity comes first. If the induced current will produce contractions in the affected muscles, it should be employed; but if, as often happens, it should fail to do so, the primary current interrupted must be brought into service. In the communication[1] already cited, I called attention to this valuable agent in the treatment of organic infantile paralysis, and adduced several cases in illustration of its beneficial action. If a contraction can be induced by it, recovery is merely a matter of time, so far as that particular muscle is concerned. As soon as the muscle is so far developed as to contract to the induced current, this latter should be employed. Every alternate day is often enough for a sitting. The time necessary for each is, of course, dependent on the extent of the paralysis.

During the period from December, 1865, to December, 1870, I treated ninety-eight cases of organic infantile paralysis. Of these, the disease was so far advanced in eleven as to render it very evident, after thorough examination, that success was out of the question. In the remaining eighty-seven, no contractions could be caused in the affected muscles by the strongest induced currents in thirty-nine; while in all of these the primary current produced decided contractions. Of the eighty-seven cases, fourteen were entirely cured; twenty-eight were greatly improved; thirty slightly improved, and the remainder—fifteen—discontinued treatment before sufficient time had elapsed to ascertain the effect. Since then I have kept no very full record of my cases, but I am enabled to state that the proportions do not vary essentially from those above stated.

---

[1] *New York Medical Journal*, December, 1865.

At the best, however, the treatment must be of long duration, and even when the muscles are entirely restored they must be reëducated to the performance of their functions. Few parents, comparatively, have the patience to wait and to devote the necessary time to doing their part of the work; unless there is a reasonable assurance in regard to these points, it is better not to undertake the case. It is not, except in recent cases, a matter of days, or of weeks, but of months, and sometimes of years.

But, even when fatty degeneration is going on, the disease may be arrested by the proper use of the direct current. Fig. 47 shows the appearance of a portion of muscle as examined by the microscope, October 21, 1866. This specimen was removed from the belly of the gastrocnemius muscle before any treatment whatever had been employed, and after the disease had existed, with gradually-advancing atrophy, for about four and a half months.

FIG. 47.

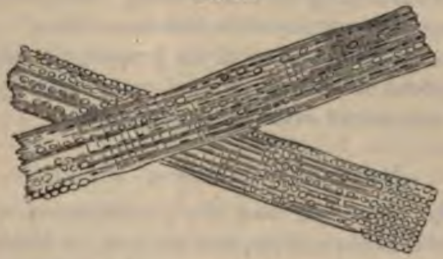
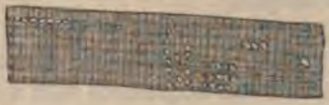


Fig. 48 represents a piece of the same muscle from the same part, on December 3d, six weeks after the treatment was begun. In the first, oil-globules are seen to have displaced the muscular tissue to a great extent; the transverse striæ have disappeared entirely from some parts, and are faintly seen even where they are present. In the second, the quantity of fat is perceived to be very much lessened, and the striæ are much more numerous and distinct. This case, which was one of paralysis of the left leg and foot, entirely recovered.

FIG. 48.



I feel that I cannot insist too strongly on the use of the primary or galvanic current, when contractions cannot be obtained by the faradaic or induced current. If the electric contractility of the muscles is not utterly destroyed—as Dr. Radcliffe[1] remarks—there appears to be no limit to the prospect of recovery.

Whichever form of current be employed, it must be applied directly to the skin over the affected muscles, or, in some cases, to the nerves which go to them; and the current should be as strong as is necessary to cause contractions. Applying it through the hand of the physician is worse than useless.

Along with the electricity, passive motions of the joints should be made, and the child should be encouraged to direct the will to the affected muscles as often and as powerfully as possible.

A very valuable aid to the treatment, in cases of deformities, is afforded by the use of apparatus calculated to take the tension from the paralyzed muscles. An overstretched muscle is in the worst possible state to react to the electrical stimulus, for the strain is of itself a most efficient agent in destroying its contractility. India-rubber cords may be very advantageously employed in this connection.

As to tenotomy, the question of its propriety must be determined by the circumstances of each individual case, and may be left to the good sense of a competent orthopædic surgeon.

b. Spinal Paralysis of Adults.

Duchenne,[2] to whom we owe the identification of several other affections of the nervous system, was the first to insist upon the fact that there was a form of paralysis met with in adults which presented great analogies with infantile spinal paralysis. He recognized two forms of this disorder, one he designated *acute anterior spinal paralysis of adults*, the other *subacute general anterior spinal paralysis of adults.* As these have the same patho-anatomical features and differ in their symptoms only as regards a few not very material points, there is nothing to be gained by considering them separately.

In the third and last edition of his great work,[3] under the head of *spinal paralysis in the adult*, he sums up his earlier and more recent investigations on the subject.

But, though Duchenne has shown by the cases recorded in the first edition of his work published in 1855, and the remarks therein made in regard to them, that he was acquainted with a form of spinal paralysis occurring in adults characterized by loss of voluntary power, atrophy, and diminished electric contractility in the muscles, "as when the anterior columns of the cord are altered," Meyer,[1] of Berlin, is entitled to the credit of being the first clearly to state in a publication his belief in the existence of an affection holding intimate relations with infantile paralysis and to employ the term spinal paralysis of adults. Thus, after describing the first named disease he says:

---

[1] Article "Infantile Paralysis." Reynolds's "System of Medicine," vol. ii.

[2] As these pages are passing through the press, the death of this distinguished physician is announced. Probably no one man has done so much as he for the advancement of neuro-pathology and therapeutics. The keenness of his observation was only equaled by his indomitable spirit of investigation and immense capacity for work. In him scientific medicine has lost a follower whose place will not soon be filled.

[3] "De l'électrisation localisée," Paris, 1872, p. 437.

"A similar paralysis of the lower extremities occurs also in adults, occasioned by the existence of some exanthematous action or other unknown cause. The disease in such cases is of course subject to such modifications as the completed structure of the body would induce. Among these are the following: 1. As the bones of the adult are fully developed, that retardation in the structural growth of the affected members, which may occur in cases of infantile spinal paralysis, has here of course no place. 2. In consequence of the adult's greater energy of will impelling him to bring into action muscles which can be made to perform the duties of the paralyzed ones, as well as in consequence of the greater firmness and resisting power of the ligaments of the adult, secondary deformities are not developed to the same extent as in the spinal paralysis of children. 3. As in no case, so far as my observation goes, is the power of locomotion removed, there cannot be so great a disturbance in the circulation of the blood, nor, consequently, so remarkable a reduction of temperature. 4. On the other hand, as a result of the double amount of work devolved upon the muscles that perform the duties of the paralyzed ones, a striking hypertrophy of these muscles is induced.

"Among other cases the following have fallen under my observation:

"The two Barons von H., twin brothers, well-built, fine large men, uniformly healthy, in their eighteenth year, simultaneously fell sick with the measles. These having run an apparently favorable course, were followed in both with a paralysis of the legs inducing a constantly increasing emaciation of those parts. When I visited them, which was not till they had reached their twenty-fourth year, the circumference of the thighs of each measured respectively twenty and twenty-one inches, the circumference of the calves ten and ten and a half inches; the latter dimension, if the normal relation of the thighs to the calves be as three to two, was accordingly four inches below the true standard. The glutei muscles, on the contrary, as the patients made all locomotory movements from the hip-joint, were developed to colossal proportions, contrasting strongly with the emaciated legs. Their walk was, therefore, very peculiar. As the legs could only be used as stilts, at every step of the right or left foot there occurred a rotary movement from behind forward of the right or left thigh, which communicated itself to the whole body, causing it to turn at every step toward the one or the other side. The extensor power of the leg was very limited; the dorsal extension of the foot and the flexion of the toes were not in the power of the patients, and but a slight adduction of the toes was possible; the patients trod upon the outer borders of the feet, and the mm. tibiales, consequently, contorted forms were exhibited. The adductors of the thigh as well as the muscles of the foot were normally developed; on the other hand, the extensors of the knee-joint and all the muscles of the leg had suffered greatly in assimilative power. The sensibility of the skin and muscles was perfectly preserved. The electro-muscular contractility was reduced in the quadriceps femoris, and altogether wanting in the mm. peronei, the extensors digit. com., the gastrocnemii, etc.; but the adductors of the knee-joint and the toes showed a weak reaction."

---

[1] "Die Electricität in ihrer Anwendung," etc., Berlin, 1868. *See* also my translation of this work, "Electricity in its Relations to Practical Medicine," second American edition, New York, 1874, p. 229.

It is, therefore, quite apparent that Meyer had a very distinct conception of the disease in question.

Since then a number of cases have been reported under different names, which are clearly instances of the affection in question, and no small amount of confusion exists in regard to the whole subject, from the fact that unnecessary refinement has been shown in classifying them. Thus, as we have seen, Duchenne describes two varieties—an *acute anterior spinal paralysis of the adult* and *subacute general anterior spinal paralysis of adults.* This latter has, by others, been designated *acute ascending paralysis.* After a full survey of the subject and careful study of several cases of each, I am very decidedly of the opinion that these two varieties do not essentially differ from each other. The affection called by Duchenne[1] *subacute diffused general spinal paralysis*—a name calculated to add greatly to the already existing confusion—is evidently *acute general myelitis.* This view relative to the identity of the two morbid states I have taught for two years past to the medical classes at the University of New York. It is sustained by very cogent reasoning by M. Petitfils,[2] and is held also with some reservation by Dr. E. S. Seguin[3] in his excellent little monograph on the affection in question.

Symptoms.—The onset of the disease is generally sudden, and is usually characterized by pains in the back, which radiate to the limbs, and by the various sensations of numbness, especially in the extreme peripheric parts of the body. There may or may not be fever, and when it is present it is not ordinarily excessive. At the same time there is loss of the power of motion, varying in character and degree from the sudden and complete paralysis of all the limbs, to the gradual extension of the akinesis from a part of an extremity to one or more. At this early period, as I have recently had an opportunity of determining, by means of Dr. Lombard's instrument for measuring differences of temperature, there is an increase of heat in the affected extremities amounting to from 2° to 4° Fahr.

---

[1] "L'électrisation localisée," troisième édition, Paris, 1872.

[2] "Considérations sur l'atrophie aiguë des cellules motrices," Paris, 1873, p. 83.

[3] "Spinal Paralysis of the Adult: Acute, Subacute, and Chronic—(Inflammation of the Motor Tract of the Spinal Cord"), New York, 1874.

From the very first and throughout the whole course of the disease, the sensibility ordinarily remains intact, and the pains which are commonly phenomena of the initial part of the primary stage disappear within the first two or three days, or even earlier, and sometimes are not present at all.

The bladder and the sphincter ani generally remain unaffected. There are usually no cramps or spasmodic contractions of any of the muscles. Neither is any feeling of constriction experienced around the body. The electric contractility of the muscles is impaired at a very early stage, and generally goes on diminishing till at last very strong induced currents fail to cause any reaction. It is rare, however, that the excitability to the galvanic current is entirely abolished, except in long-continued and neglected cases, and, even in these, currents of great intensity will often cause contractions. At the same time the cutaneous sensibility to all kinds of electrical stimulation remains unimpaired.

Reflex contractions in all the paralyzed parts are difficult, and sometimes impossible to excite from the very beginning.

The face is rarely involved. In one of my own cases, however, one side was completely paralyzed, so far as the seventh pair of nerves was concerned, and Dr. Seguin[1] has reported an instance in which the third and facial nerves were both affected. Some of the other symptoms go to show that this was not an uncomplicated case, and Dr. Seguin's diagnosis was "myelitis or degeneration of the anterior horns of gray matter of cord; the motor part being involved from the third cerebral nerve downward, with probably recent extension of myelitis to deeper parts of cord at some points."

In the majority of cases, the paralyzed parts, after a period varying from two or three weeks to several months, begin to recover their power, but it usually happens that the loss of motility remains in some muscles as in the infantile form of the disease. Atrophy may occur before the retrocession of the paralysis. Generally, however, its appearance is first seen in those parts which remain paralyzed, and occasionally it is absent altogether. In all the cases collected and observed by Seguin, it was a prominent feature; it was wanting in one of my own cases, that above referred to; as it was likewise in a very interesting instance reported by Dr. Labadie-Lagrave,[2] in which the muscles of respiration were involved, but yet in which recovery took place.

---

[1] *Op. cit.*, Case XXI., p. 19.

[2] "Observation de paralysie ascendante aiguë." Brochure, extrait de la *Gazette des Hôpitaux*, 1870.

The reduction of temperature, though marked, never, in my experience, reaches the low point observed in the infantile form. The atrophy likewise is rarely so profound. But in the case of a gentleman of New Jersey, in whom the paralysis began slowly in the left lower extremity and gradually extended upward till the medulla oblongata was involved, and death produced from asphyxia, the wasting was rapid and extensive, till at last apparently nothing of the muscular tissue remained in the limb first affected. In this case the right side continued free from the least sign of paralysis so long as the patient was under my observation.

In some cases which have been observed, the paralysis is first manifested in the lower limbs, and progressively advances upward till the superior extremities are affected. Still, in some cases continuing its progress, the medulla oblongata is reached, and death takes place by asphyxia. Or it may follow a descending course, the superior extremities being first attacked, and subsequently the inferior.

The muscles in some of these instances are very rapidly and profoundly atrophied, and can be seen to waste from day to day in groups.

Such cases may be regarded as representing the subacute form as described by Duchenne.

Other examples designated by the names of *acute progressive paralysis*, *acute ascending paralysis*,[1] *etc.*, are in reality like those described by Duchenne under the title of *subacute general diffused spinal paralysis*, and are cases of general myelitis. Of such notably is the instance reported by Harley,[2] in which the post-mortem examination was made by Lockhart Clarke, and the lesion found to implicate not only the anterior horns but the posterior, and the antero-lateral and posterior columns.

In no case that has been reported or that has come under my own notice was there any tendency exhibited to the formation of bed-sores.

From the foregoing account it will be seen that the more prominent phenomena observed in cases of spinal paralysis in the adult are strikingly like those which characterize the infantile form. Even as regards the results there is no essential variation, except that due to difference of age. There is, of course, in the adult no arrest of development, and the disposition to deformities is not so great as in the infant, but nevertheless, as in the first case reported by Charcot,[3] they may occur.

Occasionally, hyperæsthesia exists. This was the case in two of Seguin's cases[4]—XX. and XXII.—and to a marked degree in that of Labadie-Lagrave.[5] Thus, as the latter remarks :

---

[1] Landry, "Note sur la paralysie ascendante aiguë," *Gazette Hebdomadaire*, 1850, pp. 470, *et seq.*

[2] *Lancet*, October 3, 1868.

[3] "Leçons sur les maladies du système nerveux," fas. iii., 1874, p. 178.

[4] *Op. cit.*, pp. 17, 22.

[5] *Op. cit.*, p. 6.

"Besides the cutaneous hyperæsthesia, there was a still more decided muscular hyperæsthesia. The lightest pressure on the muscles was very painful, and caused the patient to cry out. In addition, lancinating pains were felt in the lumbar region, when the flexed thighs were suddenly extended. Passive movements of the lower extremities also caused a certain amount of pain."

It is very certain that many cases of spinal paralysis and atrophy occurring previously to the last two or three years, and reported under other designations, were in fact instances of spinal paralysis of adults. This is probably true, for instance, as regards the "case of acute muscular atrophy,"[1] occurring in the London Hospital in the service of Dr. Ramskill, relative to which it is stated that "electro-motility was absent," a circumstance not present in progressive muscular atrophy.

A case which forms the subject of a clinical lecture by Jaccoud[2] is clearly one of inflammation of the anterior tract of gray matter. The patient, a man seventy years of age, was seized with pains and numbness in the extremities, with incoördination. Shortly afterward there was loss of power in all four limbs, which progressively increased till at last he was unable to walk or to use his arms. At the same time atrophy began in the paralyzed parts. Reflex movements were abolished and reflex excitability was either lost or impaired, in the affected muscles. There were pains and some loss of sensibility. Death ensued: on post-mortem examination the spinal arachnoid was found studded with fibrous plates, which pressed upon the roots of the nerves, causing their atrophy. Hence the name of progressive nervous atrophy which Jaccoud gives to the case. The spinal cord was pronounced healthy, but, as no microscopical examination was made of it, the opportunity was lost for discovering the real and essential lesion, the disease of the anterior horns, which undoubtedly existed.

Some of the cases which I have, previous to the recognition of the affection under notice, regarded as instances of spinal congestion, progressive muscular atrophy, and antero-lateral spinal sclerosis, were, I have now no doubt, examples of inflammation of the anterior tract of gray matter. Several of these I have reported.

Among them is the case of Rose Peyton, who formed the subject of a clinical lecture I delivered at the Bellevue Hospital Medical College[3] in the autumn of 1870, and of which my clinical assistant, Dr. Cross, prepared at the time the following report: "Rose Peyton, twenty-seven years of age, born in Ireland, mother of two children, both of whom are living; the older has talipes valgus, while the younger is a fine, hearty child. Her family is very healthy, and there is no evidence of nervous diseases either in it or in any of its branches, so far as she is aware. The patient was a strong, active woman, and always did her own work until twelve weeks ago. In May there was a cessation of menstruation, and in July last she was seized with a deep, dull, aching pain in both legs, and which appeared to her to be in the bones. There is no syphilitic taint in her history. There succeeded, shortly after, a severe pain in the back, which has continued up to the present time, but which has varied in intensity. Soon, loss of motility, numbness, and anæsthesia, made their appearance in both legs, and in the course of two months she was totally unable to walk. At first, her bowels were very costive, but soon this condition was succeeded by incontinence of the rectum, which lasted for two weeks, varying in degree. There was also retention of urine. Sensations of formication alternating with numbness, of heat and cold, of pricking by pins and needles, were present not only in the feet and toes, but also in the hands and fingers. Patient noticed that on rising in the morning, after a night's rest, her limbs were weaker, and that she had greater difficulty in moving about. The paralysis, after commencing in the lower extremities, rapidly extended to the upper. *August 25th.*—Was able to get out of bed for the first time in five weeks, and by means of a chair could move about a very little. Since then she had improved only so much as to be able to come to the out-door department of the New York State Hospital for Diseases of the Nervous System, by being supported by a person on each side, and only then with extreme difficulty. She was admitted September 22, 1870, when she was found in the following condition: Motility and tactile sensibility in both legs greatly impaired, but the right leg is the weaker of the two. Left hand, as measured by the dynamometer, is much feebler in power than the right, and this to a more marked degree than any normal disparity. Sensations of formication, alternating with numbness, of heat and cold, pricking by pins and needles, and tingling, still continued in the feet and toes, as also in the hands and fingers. Pain in the back increased by pressure and percussion, but no burning sensation on applying heat and cold. The anæsthesia is more marked in the thighs than in the legs; soreness in the soles of the feet; bowels constipated; bladder normal; electro-muscular contractility and sensibility greatly diminished. No band around the waist. No spasms, twitchings, or reflex movements in the legs. Pain in the lower extremities as at first. Changes in the degree of paralysis from time to time. Temperature diminished. The circumference of the legs is diminished to a marked extent owing to the atrophy of the muscles. Heart and lungs healthy; urine not examined."

---

[1] Quoted from the *Lancet* in the *Quarterly Journal of Psychological Medicine*, vol. iii., 1869, p. 198.

[2] "De l'atrophie nerveuse progressive," "Leçons de clinique médicale," second edition, Paris, 1869, p. 372.

[3] "Clinical Lectures on Diseases of the Nervous System," *Quarterly Journal of Psychological Medicine*, January, 1871, p. 22.

At the time, I regarded this case as one of spinal congestion, and this was probably an associated condition, but it is very evident that it was an instance of inflammation of the anterior tract of gray matter chronic in character. The treatment by my direction consisted of electricity and ergot, and a complete recovery was the result.

The case of Elbert Baxter, detailed in the same lecture, was probably one of inflammation of the right half of the cord involving the anterior tract of gray matter and right posterior column. There were paralysis with atrophy of the right lower extremity, and marked anæsthesia and incoördination in the left. This patient also recovered after having been under treatment with ergot and electricity at the New York State Hospital for Diseases of the Nervous System for over a year.

Another case, likewise a patient in this hospital, and the subject of another clinical lecture, was at that time, February 18, 1871, regarded by me as an instance of progressive muscular atrophy beginning with congestion. It is Cases X. and XVII., of those collected by Dr. Seguin,[1] who saw the patient two days before I did, and who then considered it an example of spinal congestion. It is also briefly cited in the former editions of this work,[2] and in full in a subsequent publication.[3]

The affection began with pain in the back and sharp, shooting pains in the legs, attended with weakness. There was also, at first, some headache, vertigo, confusion of ideas, etc. Numbness and loss of power existed in both the upper and lower extremities. Subsequently, the anæsthesia and paralysis of the upper extremities disappeared. Six months afterward the head-symptoms recurred, and there were superadded fibrillary contractions in both arms and legs, with a return of the numbness. The paralysis of the lower limbs increased to such an extent, that the patient was obliged to use crutches, and six weeks later he was confined to bed, unable to move any part of his body but his head. The bladder and its sphincter were also weakened, though he did not lose control of them. The paralysis of the arms again disappeared, but it remained in the legs, and he now noticed that they began to be atrophied, and this condition went on advancing. For three years he did not walk at all, and during this time the fibrillary contractions continued in the legs, though to a diminished extent. He then gradually reacquired the power to walk with a crutch. At the time of his admission to the hospital his condition, as ascertained by Dr. Cross, was as follows :[4]

"In the legs the extensors, together with the gastrocnemii and solei muscles, were found to have almost disappeared, while the atrophy in the thigh was distinctly visible, and this loss of power had been directly proportioned to the extent of the atrophy. The gait of this patient was also highly characteristic of the disease from which he was suffering. In walking he lifted his feet high from the ground through the action of the flexors of the thigh upon the pelvis, in order to clear his toes, which drooped to an extreme degree—and his knees were in this way bent to a greater extent than usual. The legs were very much reduced in size, and the loss of muscular fibre was quite apparent from the greatly diminished electric contractility in these parts. There was no atrophy to be discerned in any other part of the body, nor did the patient have any head-symptoms whatever, nor had he any loss of motility, or any abnormal sensations in his upper limbs. His bowels were regular, and he had no trouble with his bladder. There was no loss of sensibility, nor were there any sensations of numbness in the legs. His heart and lungs were in a healthy state. The reflex excitability was diminished in the lower extremities, as was likewise the temperature, and the capillary circulation was very sluggish, as was demonstrated by the decrease of temperature, which was several degrees below the normal standard, and the effect of pressure. There were no fibrillary contractions present, nor had the patient experienced any electric-like pains, cramps, jerkings, or other abnormal sensations for some time. The outlines of the fibulæ and tibiæ, together with the knee-joints, were distinctly visible, owing to the destruction of the muscles on the anterior surface of the leg, while the posterior aspect of the calf was flattened from a like cause. His back-ache had completely disappeared, but, although he felt well and suffered no pain, he appreciated the gradual loss of power in his lower extremities. His appetite was good, and his mind was very active."

---

[1] *Op. cit.*, pp. 8 and 10. *See* note of Dr. Seguin appended to p. 11 of his Memoir.

[2] Former editions, p. 666.

[3] "Lectures on Diseases of the Nervous System," New York, 1874, p. 147—history prepared by Dr. Cross.

[4] "Clinical Lectures," p. 150.

In his recent memoir, Dr. Seguin classes this case as one of spinal paralysis of the adult, in which opinion I entirely coincide. At the time I described it, the disease under notice was not distinctly recognized, and certainly the resemblance to progressive muscular atrophy was very great. With locomotor ataxia, to which affection Charcot[1] assigns it, it has scarcely any thing in common.

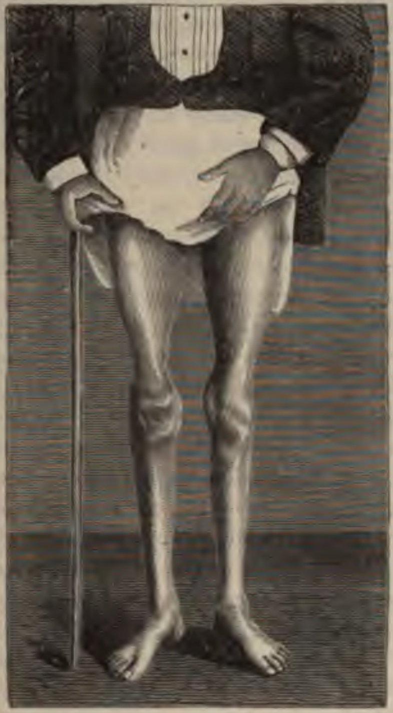
The cut (Fig. 49), owing to the position of the patient when the photograph was taken, does not show very well the effect of the disease in the legs, but the atrophy of the thighs is distinctly indicated.

Two cases which I had regarded[1] as instances of "antero-lateral spinal sclerosis" were very probably examples of inflammation of the anterior tract of gray matter. In one of these, a gentleman whom I first saw in consultation with my friend Dr. Walter F. Atlee, of Philadelphia, and who was, subsequently, for a long time under my immediate charge, the lesion was in the beginning confined to the very lowest part of the spinal cord. Gradually the disease extended upward last, after three years, the muscles of respiration and of deglu- ecame implicated, and death took place. But for several months this the patient was unable to use either legs or arms, or even to At no time, however, was the bladder deranged in any respect,

---

[1] "A Treatise on Diseases of the Nervous System," New York, 1871—and subsequent editions, pp. 475, 476.

FIG. 49.



no time were there pains or spasmodic action of the muscles. aneous sensibility was scarcely affected, and the atrophy, though ve, was not profound, and did not strike me at the time as ery active in character.

other case was that of a distinguished legal gentleman of New , sent to me by my friend Dr. Cabell, of the University of Vir- There was a gradual extension of the disease without any at- pains, anæsthesia, or muscular contractions, except to a slight at first. In this instance also the bladder and rectum escaped. se resisted all treatment. The patient finally went abroad, and on afterward in London. The atrophy was not a prominent

nother case, that of a gentleman from New Jersey, there was a condition of paralysis, involving, however, only one lateral half ody, and beginning in the leg. In this case the atrophy was of the most active character, advancing *pari passu* with the paraly The flexors and extensors of the foot, and the flexors of the leg, w almost entirely destroyed when the patient came under my obser tion. Before I saw him, however, he had consulted several distinguis medical gentlemen, who had treated his case as one of tumor of cord or of the vertebral column. This case has already been cited page 474, and is noticed in the previous editions of this work.[1]

In regard to these three cases, I stated in 1871,[2] "Such cases as foregoing, and several others which have come under my notice, ar doubtless to be classed with many of those placed under the head of what Duchenne has called general spinal paralysis."

Since 1873 I have had the opportunity of witnessing many cases of spinal paralysis of adults. Some of the more striking of these will be noticed under other divisions of this section.

Causes.—In many cases of spinal paralysis of adults, the disease is clearly the result of cold, either applied directly to the back as in lying on cold, damp ground, or from refrigeration of some part of the surface of the body. Relative to this last influence, Frinberg[3] has performed an experiment which, if confirmed in its results, will be of a very instructive character. He shaved off the hair from the skin of a rabbit and on the unprotected skin threw a jet of the vapor of ether by means of Richardson's apparatus. Three days subsequently he repeated this operation. About a month afterward the animal was attacked with incontinence of urine and paraplegia, and died in a few days. On post-mortem examination the whole length of the spinal cord was found inflamed. There was in fact general acute myelitis. In regard to this experiment, I can adopt the language of Vulpian,[4] who says:

"This experiment would be very valuable if the results obtained had been observed with a certain number of other animals treated in the same manner. Till then we may be permitted to doubt if there really was the relation of cause and effect between the refrigeration of the skin by the ether-spray and the paraplegia which made its appearance a month later."

Bernhardt's[5] case ensued upon exposure to cold, as did several of Seguin's, and five in my own experience. Meyer's[6] two cases followed close on measles. In Rose Peyton, whose case I have related, sudden suppression of menstruation appeared to be the cause; in a number of others, blows and falls were alleged as causes, and in others venereal excesses, dysentery, syphilis, and violent muscular efforts, seem to have been the exciting agencies. In the majority of cases, however, no cause can be discovered. Such at least has been the fact with the instances that have come under my own observation.

---

[1] *Op. cit.*, p. 476.

[2] Previous editions of this work, p. 476.

[3] "Ueber Reflexlähmungen," *Berlin. klin. Wochenschrift*, 1871, Nos. 41, 42, 44, and 45.

[4] "Leçons sur l'appareil vaso-moteur," Paris, 1869, tome ii., p. 88.

[5] "Ueber cime der Spinalen-Kinderlähmung ähnliche Affection Erwachsener," "Archiv für Psychiatrie und Nervenkrankheiten," B. iv., 1873.

[6] *Op. cit.*, p. 229.

Diagnosis.—Spinal paralysis of adults is to be recognized by the facts that the paralysis is often extensive in the first place, and then becomes restricted, or that it begins in a limited portion of the body, usually in one or both of the lower extremities and then advances; that the paralysis always precedes the atrophy; that the reflex excitability is somewhat impaired; that the electro-muscular contractility is diminished; that there are no bed-sores; that the disturbances of sensibility are not usually prominent features; and that the bladder and rectum generally escape.

It has been often confounded with progressive muscular atrophy, but attention to the features above stated will prevent mistakes of the kind. In progressive muscular atrophy, it must be borne in mind that the atrophy is the essential feature, and that the loss of power results from the diminished size of the muscles.

In acute general myelitis the paralysis of the bladder and sphincter ani, the tendency to bed-sores, the spasmodic movements of the limbs, the great disturbances of sensibility, the sensation of constriction around the body, and the greater constitutional commotion, will serve for the identification of the disease. In the partial form of acute myelitis the distinctive features are equally as marked.

Hallopeau[1] has reported a number of cases under the head of *chronic diffused myelitis*, which were undoubtedly instances of spinal paralysis of adults, judging both from their symptoms and morbid anatomy, and the author admits as much when he says:[2]

"The remarkable lesions [brown discoloration, no microscopical examination being made] which we found in the anterior horns permit us to think that, as in the cases of MM. Charcot and Joffroy, histological alterations would have been discovered."

The distinction between the acute, the subacute, and the chronic forms of spinal paralysis of adults is not one of kind but only of degree, and the same may be said of the acute ascending paralysis of Landry, on which I have already insisted. The fact that in the latter form of the disease the respiratory muscles are affected, is of course only due to the circumstance that the morbid process has reached the medulla oblongata. In regard to this variety, Dr. Seguin says: "There is an affection running its course in ten or twenty days, characterized by symptoms almost identical with those of subacute spinal palsy. There is an akinesis, without much anæsthesia, first appearing in the feet and legs, then ascending and involving the entire trunk and limbs, producing, in nearly all cases, death by asphyxia. It is upon this palsy of the respiratory muscles that the diagnosis of this most fatal disease, acute ascending paralysis, is to be made from spinal paralysis."

---

[1] "Études sur les myélites chroniques diffuses," *Archives Générales*, 1871-'72.

[2] *Op. cit.*, tome i., 1872, p. 72.

Now, if in any case the progress of the disease had been arrested at a point of the spinal cord half an inch below the decussation of the anterior columns, the diagnostic mark of Dr. Seguin would have been absent, and the distinction between the form in question and spinal paralysis of adults could not have been made. The mere fact of the implication of the respiratory nerves cannot, in my opinion, be made a ground for assuming a separate nosological position for acute ascending paralysis any more than the circumstance of the brachial plexus being reached in any case should make a distinct form. Dr. Seguin does not appear to recognize the fact that the acute ascending paralysis of Landry is identical with the subacute general anterior spinal paralysis of adults, although he very distinctly admits the relationship.

Petitfils[1] has entered at length into the consideration of the question of the identity of the acute and subacute forms, and has very satisfactorily shown, both from the symptoms and the morbid anatomy, that no essential difference between them exists.

From spinal congestion the spinal paralysis of adults is discriminated by the facts that, in the former the sphincters are usually affected, that the paralysis is not generally complete in any part of the body, by the absence of atrophy, and by the general presence of disturbances of visceral functions. In the first stage, however, of either affection, it must be admitted that very striking resemblances exist, and time may be necessary for the diagnosis to be made with accuracy. Thus, in a case which I saw a few days since, in consultation with Dr. Newcomb of this city, there had been, in the first place, a set of symptoms present which, had I then seen the patient, would have induced me to regard it as one of spinal congestion. When the man, a stage-carpenter, came to me, however, the paralysis and atrophy of the right lower extremity, the diminished temperature of that limb, and the absence of bladder-troubles, left no doubt on my mind relative to the case being one of spinal paralysis in the adult.

A case reported by Dr. Cuming,[2] of Belfast, which presented all the essential features of spinal paralysis of adults, was regarded by him as one of spinal congestion. The patient, a man aged forty, observed on a cold night that his hands had become numb and white, and when he reached home he had not the use of them. A few days afterward he fell asleep on a cold wall, and when he awoke found the numbness increased. In a few days he was entirely deprived of the power of motion in all parts below the neck. But he soon began to regain the use of his limbs, and at the end of two years could walk well. The upper extremities were, however, wasted, and he had the *main en griffe.*

---

[1] "Considérations sur l'atrophie aiguë des cellules motrices," Paris, 1873, p. 83.

[2] "Case of Extensive Paralysis from Morbid Condition of the Spinal Cord, probably Congestion," Transactions of Ulster Medical Society, *Dublin Quarterly Journal of Medical Science*, vol. xlvii., 1869, p. 471.

The diagnosis, therefore, should not be hastily made.

Prognosis.—So long as the lesion does not attain to the height of the respiratory nerves, the prognosis, as regards the life of the patient, is not unfavorable. Indeed, recovery, with a more or less extensive loss of power, with atrophy and deformity, is the rule, and in some cases there is a complete restoration of motor power and muscular integrity. Even when the morbid process reaches the height of the respiratory nerves, life may be preserved, and complete restoration may take place. This was the case with the instance already cited reported by M. Labadie-Lagrave, and in two in my own experience, which will be fully cited under the head of treatment. Sometimes the process of recovery begins within a few days, and goes on uninterruptedly till complete restoration is the result.

When seen at a later stage, when the paralysis and atrophy are limited, the prospect of cure or improvement depends altogether on the condition of the muscles as regards their electric contractility. If the affected muscles can be made to contract with either the induced or primary current, recovery will, in all probability, take place. But, when this action cannot be brought about, there is no hope. The principles of the prognosis are, therefore, identical with those which exist in the infantile form of the disease.

Morbid Anatomy and Pathology.—There is not much to add under this head to the remarks made on the same subject in regard to infantile spinal paralysis. The characteristics of the disease have, as we have seen, sufficed to place the lesion in the anterior tract of gray matter, and this theory, based upon physiology and the analogy of the affection with infantile spinal paralysis, has been definitely confirmed by post-mortem research within the past three years by Gombault,[1] one of the pupils of the Salpêtrière.

The patient, aged sixty-seven on the 1st of January, 1865, was seized suddenly with a paralysis of all four extremities, beginning in her legs and extending to the arms as a numbness and heaviness. Within half an hour she could not stand. There were no antecedent phenomena, she having been in perfect health up to the moment of the attack. There was no paralysis of the tongue, muscles of deglutition, or respiration. The bladder and rectum were also unaffected, and the cutaneous sensibility remained intact.

The paralysis of the limbs soon became complete, and in fifteen days she was taken to the hospital. There was slight febrile disturbance, but at no time were there bed-sores.

---

[1] "Note sur un cas de paralysie spinale de l'adulte suivi d'autopsie," *Archives de Physiologie*, tome v., 1873, p. 80.

After two years passed in complete immobility, the patient rec[illegible]ered, to some extent, the use of her limbs. The amendment began the upper extremities. When she entered the Salpêtrière, five [illegible] a half years after the inception of the disease, she could walk imp[illegible]fectly with a cane. During the first year of her stay in the hospital improved so that she was able to dress herself, and to take short w[illegible] in the court-yard.

On the 13th of May, 1872, examination showed that the thenar [illegible]nences had entirely disappeared, the interosseous muscles were a[illegible]tr phied, there was the *main en griffe ;* the muscles of the forearms, a[illegible]rms shoulders, neck, and chest, were atrophied.

In the lower extremities the left calf was most atrophied, and was soft and flabby ; the thighs were unaffected.

The electro-muscular contractility was entirely abolished in the hands and forearms, impaired in other parts of the upper extremities, and in the legs. The cutaneous sensibility was preserved.

The patient soon afterward died of another disease.

On post-mortem examination the membranes of the brain and cord were found to be healthy, and to the naked eye there was no lesion of either of these organs.

The histological examination of the spinal cord was made after hardening in solution of chromic acid and coloring with carmine.

The white substance throughout all its extent exhibited no traces of disease. Only the columns of horizontal fibres which emerge from the anterior horns to form the fibres of origin of the anterior roots showed a notable diminution in size. The posterior commissure and posterior horns were normal. The lesion was almost entirely confined to the area of the anterior horns, and here it only concerned the large nerve-cells called motor-cells. The walls of the vessels had suffered no change; they were of normal thickness, and the sheath was free from granular bodies. Moreover, there was not in the neuroglia any trace of the existence of an irritative process such as a proliferation of the neuroglia.

As to the alteration of the nerve-cells, it was such as is met with in progressive atrophy of these elements—yellow pigmentation. The lesion was diffused; it had struck here and there the nervous elements, of which a certain number had disappeared, for in some sections only fifteen or twenty could be counted.

The cells which did not exhibit this yellow pigmentation were nevertheless reduced in size.

This was the first full investigation made relative to the morbid anatomy of spinal paralysis of adults; but, previous to Gombault's researches an examination of a patient who had died of ascending paralysis, and in whom lesions of the anterior horns were discovered, was reported by Chalret.[1]

---

[1] "Thèse de Paris," 1872, cited by Gombault.

The data are, therefore, quite sufficient to enable us to place spinal paralysis of adults in a definite patho-anatomical position as depending upon inflammation of the anterior tract of gray matter and the consequent atrophy and disappearance of the cells constituting its nervous elements.

In regard to the questions entering into the pathology of the disease under notice there is nothing to bring forward in addition to the facts and arguments already adduced under the head of infantile spinal paralysis.

Treatment.—The treatment of spinal paralysis of adults admits of division into two parts, that which is proper for the first or acute stage, and that advisable for the second or chronic stage.

I have had the opportunity of treating four cases of the disease in question from the very beginning, with the result in each case of arresting the progress of the disease and preventing any subsequent atrophy of the limbs. Two of these were of the most severe type of this affection, and I therefore report them with some degree of fullness, as exemplifying the therapeutical principles which in my opinion should govern.

A. G. S., aged about thirty-five, after rising one morning and moving about the room, felt a slight degree of weakness in both lower extremities. This increased through the day, and by night he was unable to stand. The next morning he felt similar weakness in both arms and in a few hours was deprived of their use. He was out of the city at the time, but he was brought here, and I saw him on the fourth day. He was then perfectly helpless from complete paralysis of all four limbs. There were no aberrations of sensibility, no paralysis of the bladder or sphincters, no motor spasms anywhere. Reflex excitability was abolished in all the paralyzed parts, and the electro-muscular contractility was greatly diminished especially in the muscles of the legs. The breathing, deglutition, articulation, and motility of the neck and face, were unaffected. The mind was as clear as ever. There had been slight fever, but this had disappeared when he came under my observation. There was no history of syphilis.

I immediately began the treatment with the iodide of potassium in doses of ten grains three times a day, increased gradually, and the fluid-extract of ergot in doses of a drachm, to be taken also three times a day.

On the following morning there was some difficulty of respiration and of deglutition, and the movements of the tongue were a little awkward. The irregularity and shortness of breathing increased through the day and night, and when I saw him the next morning there was great discomfort on this account. The action of the heart was also considerably disturbed, and there were frequent interruptions in the pulse. On the seventh day of the disease he suddenly became paralyzed on both sides of the face, the right being more severely affected.

During all this time the iodide of potassium and ergot had been persistently given, the latter, on the appearance of the bulbar symptoms, having been increased to two drachms four times daily.

On the ninth day of the disease there was a slight amelioration in the phenomena due to the implication of the medulla oblongata. The respiration became easier, the deglutition less difficult, the articulation more distinct, and the facial paralysis of the left side began to disappear. He was able to close the eye of that side and to elevate and corrugate the brows.

On the tenth day the facial paralysis of both sides had nearly disappeared, and the patient was able to breathe freely, to talk well, and to swallow without inconvenience. There was also a slight return of motility in the lower extremities. The toes could be moved and the feet flexed.

The galvanic current, interrupted rapidly, was now applied to the muscles of both upper and lower extremities for half an hour every day, at the same time that the internal medication was continued. The limbs were also well kneaded, and passive motions made with them frequently.

On the thirteenth day his condition was as follows : He could move both lower extremities while lying in bed—performing with slowness, but yet with precision, all the movements of which the parts were capable. The arms could not yet be moved, but he could slightly extend and flex the fingers of both hands. The bulbar symptoms had entirely disappeared. Reflex excitability and electro-muscular contractility were good, except that it required strong galvanic currents to cause contractions in the anterior tibial and peroneal muscles of both legs. All the other muscles reacted well to the faradaic current. The ergot and iodide of potassium were now discontinued.

His improvement went on, and by the end of the fourth month he could walk a mile or more, and use his hands and arms well. There was slight atrophy of the muscles of the calves, but nowhere else. The faradaic current was still employed daily, and under its use he became stronger, till at the end of a year he was not conscious of any weakness in any part of his body. He has continued and now is perfectly well, and was kind enough to allow me to make him the subject of a clinical lecture a few days ago at the University Medical College.

B. B., aged forty-five, was attacked with gradually-increasing paralysis of the right side, beginning in the leg, and gradually advancing during several months, till it involved the whole of the lower extremity and arm. At no time, however, was the loss of power complete. He went to the Warm Springs of Arkansas, but did not improve. Returning to New York in April, 1875, and his disease becoming worse, I was requested to take charge of his case.

When I saw him there was such a degree of paralysis of the right lower extremity that he was unable to walk without assistance—the arm of that side was nearly useless. The respiration was labored and irregular; he was almost unable to swallow, and would not eat, on account of the great distress produced by all attempts at deglutition—the tongue could not be protruded, and his articulation was unintelligible. Owing to his inability to swallow, the saliva ran in streams from his mouth, and, as he could not cough without great and painful effort, the mucus accumulated in his air-passages, and caused danger of suffocation. It was removed from time to time from the pharynx by the fingers of his nurse. There was moderate febrile excitement.

Although the paralysis was more marked on the right side, I ascertained that the left was also affected. Tickling the soles of the feet caused no reflex movements. Electro-muscular contractility was greatly impaired on the right side, and weakened quite notably on the left. There was no facial paralysis; no bladder or sphincter trouble; no bed-sores; no derangement of sensibility; no pains, and no muscular spasms.

At no time had there been any mental disturbance, except great emotional weakness and irritability of temper. The intellect was perfectly intact; the memory perfect.

The iodide of potassium was given as in the previous case, but was combined with the bromide in doses of fifteen grains. Ergot, in the form of the fluid-extract, was also administered. I requested my friend Dr. Clinton Wagner to make a careful examination of the throat, and to take charge of him, so far as the immediate management of his throat-symptoms was concerned. He found the fauces, pharynx, and larynx congested, and the vocal cords partially paralyzed. He recommended steam inhalations, and they were used, with the effect of giving great relief by detaching the mucus and rendering it more fluid.

As the difficulty of swallowing increased, I made preparations to feed the patient through a stomach-tube. The efforts at respiration became more painful, and at times I thought death by asphyxia imminent. The tongue was now immovable, lying like a flabby, reddened mass in the mouth, and the patient lay in bed entirely helpless through the paralysis of his limbs. But now amendment began, and, as in the case just cited, with the gradual disappearance of the bulbar symptoms. Little by little improvement took place. Faradization was now brought into use, and was employed daily to the tongue, throat, and extremities, while the internal medication was continued. By the first of June he was able to use his legs in standing, and his arms and hands to support himself. He could not yet, however, employ them in feeding himself. About the first of July he could walk with a cane, and used his hands well. He went to Saratoga the middle of July, and while there had a relapse, consisting in a sudden paralysis of the left lower extremity, by which he was again deprived of the ability to walk. He was there attended by Drs. Whiting and Lente, and I also visited him. The iodide of potassium, which had been discontinued, was resumed. Under its use, with ergot, hypodermic injections of strychnia, and faradism, he has again acquired the power of walking, though his improvement, owing to considerable atrophy of the muscles of the legs, especially the gastrocnemii, is slow.

It may be mentioned, incidentally, that after he began to lose power in his legs, he fell, upon one occasion, and struck his right side violently against the edge of a wooden bucket. After he was able to go out, I made a careful examination, and, detecting fluctuation in the liver, I removed about a pint of pus with the aspirator. No unpleasant symptoms followed, and there was no reaccumulation of the pus.

The treatment, therefore, which in my opinion is best adapted to the initial or advancing stage of spinal paralysis in the adult, is that which consists in the persistent use of the iodide of potassium and ergot, both given in large doses. The former I carried, in both of the cases cited, to half an ounce daily, and the latter to an ounce. Dr. Seguin[1] reports a case, as occurring in the practice of Dr. T. A. McBride, and which he saw in consultation, in which the fluid-extract of ergot was given in like quantity daily, and in which recovery ensued. This treatment is based upon the theory that the first stage of the disease in question is characterized by a congestion limited to the anterior tract of gray matter.

As soon as the muscles show the slightest sign of regaining their power, electricity should be employed. The form in which it should be used depends entirely on the requirements of each individual case. If the faradaic current will cause contractions in the paralyzed muscles, it is the preferable form, but if not, then the interrupted primary or galvanic current must be applied and used in such a degree of intensity as will cause muscular contractions.

In one of the other cases of the four which I have treated, while the disease was advancing, I used the actual cautery to the spine—applied over the seat of the disease, as near as could be determined from the extent of the paralysis. The effect was apparently excellent, the lesion ceasing to advance. But one such case cannot be regarded as affording more than an indication. From what I have seen, however, of the power of the actual cautery in other affections of the cord, I should be disposed to employ it in future like cases of spinal paralysis of adults. In the later or chronic stage, as will be presently shown, it is certainly of great value.

After the progress of the disease is arrested, the treatment which is most advisable consists in the persistent use of electricity to the paralyzed muscles, with the view of restoring motility and preventing or curing atrophy; the hypodermic injections of strychnia in gradually-increasing doses, till the physiological effects of the drug are produced, when the doses should be diminished, and again increased, and so on; and repeated applications of the actual cautery to the spine.

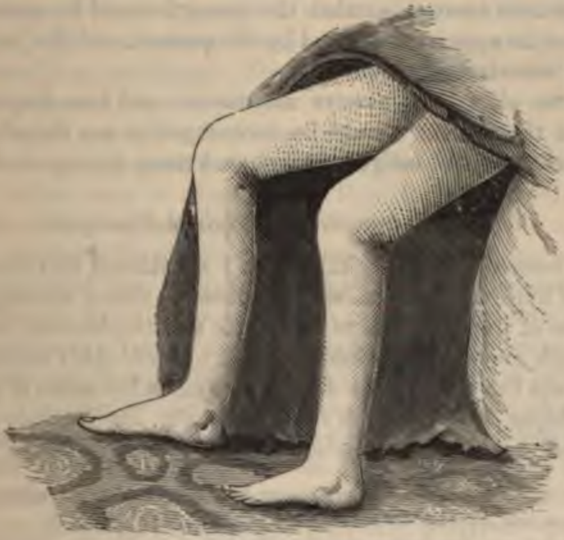
---

[1] *Op. cit.*, p. 22, Case XXII.

Relative to this latter agent, my method consists in first applying the ether-spray over the part to be cauterized, and using the platinum-tipped iron brought, by means of an alcohol-lamp, or a Bunsen's burner, to a white heat. Three or four applications are made at one sitting on each side of the spinous processes, and over the part which is in physiological relation with the paralyzed regions.

I have never seen a case of spinal paralysis of adults which was entirely unamenable to this treatment, and the majority recover completely. In the accompanying woodcut (Fig. 50) is the exact appearance of the legs of a woman who consulted me September 20, 1874, and who had suffered an attack of the disease under consideration some three years previously. As will be seen, the calves are atrophied to an extreme degree, and her walking was correspondingly impaired. She was treated with the galvanic current in the first place, and subsequently with the faradaic. Strychnia was injected into the limbs daily, according to the method mentioned, beginning with the thirtieth of a grain, and the actual cautery was applied to the lower dorsal and upper lumbar region of the spine six times. In less than three months she could walk as

FIG. 50.



well as she ever did, and her calves, from having measured each only eleven and a half inches at their largest circumference, had increased to fifteen inches in the right, and fifteen and a half in the left.

Electricity has been very generally employed by those physicians who have recognized the disease in question. Thus Bernhardt[1] reports a case of recovery mainly through its agency, as do also Eisenlohr,[2] a case from Friedreich's clinic; Frey,[3] three cases from Kussmaul's clinic in Freiberg; Seguin,[4] several cases, in which electricity was a part of the treatment, and with good results; Lincoln,[5] complete recovery after marked atrophy; Leyden,[6] a bad case with partial recovery, so as to be able to walk with crutches a little better than he could before treatment; and cases mentioned by Duchenne.[7]

In my own practice, I have treated a good many cases with electricity alone—cases in which the paralysis and atrophy were limited, and have rarely been disappointed in the results. In one very notable case, sent to me by my friend Dr. Christopher Johnston, of Baltimore, the gastrocnemius was rapidly regenerated through the agency of the interrupted galvanic current, so that the strength could be measured daily by means of an apparatus devised by the patient, and the improvement accurately ascertained.

With the electricity, passive movements and kneading are always useful, and the patient should be encouraged to use the affected muscles up to the point of fatigue, at repeated times during the day.

c. Pseudo-hypertrophic Spinal Paralysis.

In the former editions of this work I considered this disease under the head of hypertrophy of muscular connective tissue, although treating of it as one of the affections of the motor and trophic cells of the cord.

Although previously noticed, the first to thoroughly investigate the condition was Duchenne,[8] who described it under the name of *paraplégie hypertrophique de l'enfance de cause cerebrale.* He has since designated it *paralysie pseudo-hypertrophique, ou myo-sclérosique.*[9] Jaccoud[10] calls it *sclérose musculaire progressive* (progressive muscular sclerosis). Dr. Foster[1] terms it paralysis with apparent muscular hypertrophy, and Barth[2] fatty muscular atrophy.

---

[1] "Ueber eine der spinalen Kinderlähmung ähnliche Affection Erwachsener," *Archiv für Psychiatrie und Nervenkrankheiten*, Band iv., Heft 2, 1873, p. 370.

[2] "Zur Lehre von der acuten spinalen Paralysie," *Archiv für Psychiatrie u. s. w.*, Band iv., 1874, p. 219.

[3] "Ueber temporäre Erwachsener, die der temporären Spinallähmung der Kinder analog sind, und von Myelitis der Vorderhörner auszugehen scheinen," *Berliner klinische Wochenschrift*, Nos. 1–3, 1874.

[4] *Op. cit.*, Cases XIX., XX., XXII.

[5] "A Case of Spinal Paralysis in an Adult, resembling the so-called Infantile Paralysis," *Boston Medical and Surgical Journal*, March 25, 1875.

[6] "Klinik der Nervenkrankheiten," zweiter Band, Berlin, 1875, p. 199.

[7] *Op. cit.*, p. 458.

[8] "De l'électrisation localisée," etc., Paris, 1861, p. 353.

[9] *Archives Générales*, etc., 1868.

[10] *Op. cit.*, p. 365.

Regarding the affection as consisting essentially in disease of the motor and trophic nerve-cells, and as being manifested by hypertrophy of the muscular connective tissue at the expense of the muscular fibres, I have provisionally placed it in the present chapter. My personal acquaintance with the disease is limited to six cases, and I am of the opinion that it is exceedingly rare in this country—eleven other cases only having been reported: one by Drs. William Ingalls and S. G. Webber, of Boston,[3] the latter of whom, in connection with the history of the case, has written a very excellent memoir on the disease; one by Dr. William Pepper,[4] of Philadelphia; one by Dr. S. Weir Mitchell;[5] one by Dr. C. H. Drake;[6] one by Dr. C. T. Poore,[7] of New York; four by Drs. Steele and Kingsley,[8] of Missouri; and two by Dr. George S. Gerhard,[9] of Philadelphia. The American cases known to me are thus seventeen in number.

Symptoms.—The first symptom observed is weakness in the lower extremities, which causes an inability to stand steadily, or to walk without stumbling or falling. The legs are separated widely in standing or walking, and thus a peculiar character is given to the gait, which somewhat resembles that of a duck.

Very soon an enlargement of the calf of one of the legs is perceived, the other before long is affected, and then the muscles of the thighs and gluteal region become involved.

As the child stands or walks, a remarkable incurvation of the spine in the lumbo-sacral region is perceived, so that if, as Duchenne remarks, a plumb-line be allowed to fall from the most posterior part of the spinous process of a vertebra, it passes far behind the sacrum. He considers this phenomenon to be due to weakness of the erector muscles of the spine. The muscles of the trunk may become involved, as may also those of the upper extremities—the deltoids being the first affected in the majority of cases, and the progress being much slower than in the lower extremities.

With the advance of the hypertrophy the paralysis becomes more strongly marked, and finally the child is confined to the recumbent posture. Distortions from disturbance of muscular equilibrium may take place, and the attempt at flexion or extension becomes painful.

---

[1] *Lancet*, May 8, 1869.

[2] "Beiträge zur Kenntniss der atrophia musculorem lipomatosa," *Archiv der Heilkunde*, 1871, p. 120.

[3] "A Case of Progressive Muscular Sclerosis, with a Paper on the same," *Boston Medical and Surgical Journal*, November 17, 1870.

[4] "Clinical Lecture on a Case of Progressive Muscular Sclerosis," Philadelphia, 1871. Also *Philadelphia Medical Times*, June 15 and July 1, 1871.

[5] *Photographic Review*, October, 1871.

[6] *Philadelphia Medical Times*, August 29, 1874.

[7] *New York Medical Journal*, June, 1875.

[8] *Philadelphia Medical Times*, October 16, 1875, p. 29.

[9] *Alienist and Neurologist*, January, 1880.

Occasionally the skin over the affected parts presents a peculiar mottled appearance, such as would be produced in the healthy skin by exposure to cold.

After a period which varies in duration from two to five or six years, the hypertrophied limbs may begin to diminish in size, and eventually they put on very much the appearance exhibited in infantile spinal paralysis. This does not appear to be a constant occurrence, but is markedly exhibited in a case now under my care. Sometimes the muscles which are attacked, as the disease advances from the lower extremities, do not become hypertrophied, but on the contrary diminish in volume as in infantile spinal paralysis. We thus have in the same individual, some muscles paralyzed with coexistent hypertrophy, while others are paralyzed and atrophied.

Electric contractility is always lessened to the induced current, but, according to some observers, is increased to the primary current. In the cases under my care, both currents failed to cause the normal amount of contraction in the affected muscles. The cutaneous sensibility is not affected.

The course of the disease is slow, its average duration being about five or six years. As it advances, there are symptoms indicating loss of mental power, and cerebral disturbance is sometimes also indicated by ocular troubles and pain in the head.

Death takes place by the respiratory muscles becoming implicated, by exhaustion, or by some intercurrent affection.

Weir Mitchell, in the Philadelphia *Photographic Review*, for 1871, reported a case which has recently been reëxamined by Dr. George S. Gerhard.[1] The most remarkable feature of the case, that of a boy now thirteen years old, is that the tongue and all of the facial muscles, but particularly the temporals, are hypertrophied. His speech is altered from the enlargement of the tongue, and he has some difficulty in taking his food. There is also a somewhat more than normal cardiac impulse. As regards the hypertrophy of the facial muscles this case is remarkable, and would be unique, but for the occurrence of a like condition in a case of my own, in which the left side of the face is hypertrophied.

In the case which came under my notice March 7, 1871, the patient, a boy seven years old, exhibited great disinclination to learn to walk. At three years of age he could not stand longer than a few seconds, and even for this time he was obliged to spread the legs apart and to hold on to some article of furniture. It was not noticed till he was five years old that his legs were larger than was natural. The hypertrophy egan in the right calf, then attacked the left, and then the glutei mus-es, before affecting the muscles of the thighs. The upper extremities e as yet unaffected, but the spinal curve is very evident. The accom-anying woodcuts (Figs. 51 and 52) give a posterior and profile view f this boy, from photographs. He was unable to stand alone while e photographs were being taken, but the spinal curve is well shown,

---

[1] "Pseudo-hypertrophic Paralysis," *Philadelphia Medical Times*, October 16, 1874, p. 31.

Fig. 51.

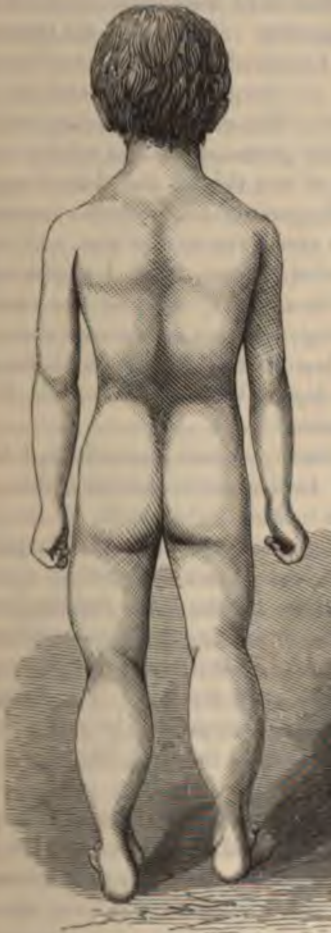
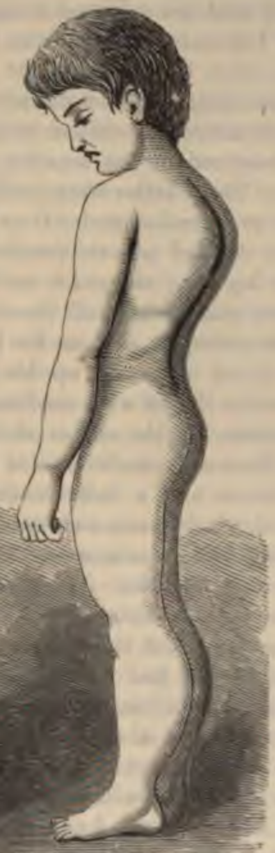


Fig. 52.



nd the positions are those he spontaneously assumed. He died in the pring of 1875, with pneumonia, having been for the previous three ears unable to stand or even sit. The muscles of the upper extremi-ies were paralyzed for two years before his death, but underwent apid atrophy instead of enlargement.

Another case, that of a bright, intelligent boy, six years of age, was brought to me May 3, 1871, at the suggestion of my friend Dr. Trask, of Astoria, who accompanied the patient. Several months previously the child had been noticed to fall frequently while at play in the house, and to show weakness in the legs when ascending a staircase. The parents were unable to account for this debility, for, as the father assured me, the legs were exceedingly well developed. As the boy stood in my consulting-room, I observed that he separated his legs to a greater than usual distance, and that as he walked he also kept them far apart, and that his gait was staggering. As soon as his trousers were removed, I at once perceived the nature of his disease, for the calves of both legs were hypertrophied to an enormous extent, and the incurvation of the spine was well marked. The electro-muscular contractility was almost entirely abolished in the gastrocnemii and solei muscles, and notably lessened in the muscles of the thighs, the gluteal region, and the back. These latter were not hypertrophied. On the contrary, they appeared to be rather under than above the normal size, and they were in a very decided paretic condition. Thus, when I requested him to cross one leg over the other as he sat on a chair, he was unable to do so without seizing hold of the leg with his hands, and thus assisting with their strength, and, as he lay at full length on his back on the floor, he could not draw up his legs without great trouble, though he could flex the thighs with readiness.

On measuring the calves at their greatest dimensions, I found the right to have a circumference of twelve and a quarter inches, and the left of eleven and a half inches. The right thigh, at its point of greatest circumference, measured but eleven and a quarter inches, and the left ten and three-quarters inches. I saw this patient again in the course of two months. The paralysis of the lower extremities had increased to such an extent as to cause walking to be very difficult. At every step he lifted the thigh almost to the line of a right angle with the body, for he had no power to raise the foot. The flexors of the thigh, upon the pelvis, did not therefore appear to be much weakened. The calves were of about the same size as before. The upper extremities were still unaffected.

I did not see this case again for nearly two years. The paralysis had then so far extended as to render walking impossible, but the arms were still strong, and by their means the patient dragged himself along over the floor. The calves had diminished in size, and the extensor muscles of the foot had become atrophied to such an extent as to allow of the permanent elevation of the heels by the uncompensated action of the still incompletely-paralyzed gastrocnemii and solei. The thighs were now hypertrophied, as were also the glutei muscles.

Accurately measured, the circumference of the calves was, for the right, eight and a half inches, a loss of three and three-quarters inches; and for the left, eight and a quarter inches, a loss of three and a quarter inches. On the other hand, the right thigh measured, at its largest part, fifteen inches, an increase of three and three-quarters inches; and the left fourteen and three-quarters inches, an increase of four inches.

I saw this patient again in the summer of 1874, a year after the last visit, when, in order to allow of his wearing a shoe, I divided the right tendo-achillis, with the result of bringing down the heel and permanently relieving the extreme condition of talipes equinus which existed. The calves had undergone still further atrophy, and the thighs were likewise beginning to shrink. There was a slight disposition to a contraction of the flexors of the thighs, and the upper extremities were becoming paretic.

A year subsequently (July, 1875) I again saw this patient. He had then been using a steel apparatus, which enabled him to stand, or rather the apparatus stood, and, being strong, supported the completely-paralyzed patient. The calves now measured, the right eight inches, and the left eight and a quarter inches in circumference—a loss from the first measurements of four and a quarter inches and three and a quarter inches respectively. The thighs had also lost greatly from their hypertrophied condition of two years before. The right now measured, at its largest part, ten and a half inches, a loss of five inches, and the left ten inches, a loss of four and three-quarters inches.

The upper extremities were decidedly weaker than they were a year ago, but there was as yet no hypertrophy. The patient could not even sit without support, and there was notable weakness of the muscles which maintain the erect position of the head.

Throughout the whole of the period during which this patient has been under my observation, the mind has remained clear, and the general health has been excellent, circulation, respiration, digestion, and urination, all being well performed. The cutaneous tactile sensibility and the sensibility to pain have not been in the least weakened.

While these pages are going through the press, I have again (December 2, 1875) examined this patient, whose general health began to show signs of giving way. To my astonishment, a feature presented itself which thus far is entirely exceptional. A second stage of hypertrophy is going on ; the calves now measure, the right ten inches, and the left ten and a quarter. The thighs were not measured, but were very considerably larger than when I last saw them ; and the father, a very intelligent gentleman, said that the enlargement in the lower extremities had been going on for two or three months. The left side of the face was decidedly larger than the right. The patient was still unable to walk, stand, or sit alone, but was comparatively strong in the arms, and in good general health. His mind was remarkably bright.

At all my examinations except the last two, I removed, by means of Duchenne's trocar, portions of the hypertrophied and atrophied muscles, the results of the examination of which will be given under the head of the morbid anatomy.

Quite recently Dr. E. B. Richardson, of Mount Sterling, Kentucky, has given me the details, with photographs, of an interesting case of the disease under notice. The patient, a boy, is eight years of age, of average intelligence, though not capable of prolonged mental exertion. The disease is of several years' duration, and he is slowly getting worse. His locomotion is peculiar; usually he walks with his hands grasping the front of the thighs, and his legs are drawn up suddenly as if with strings fastened to his back. In ascending a staircase, he does so with his hands on his thighs, and the same foot is always advanced first, and not each alternately. If sitting down, he raises himself by clasping the thighs strongly; otherwise he cannot get up at all.

FIG. 53.

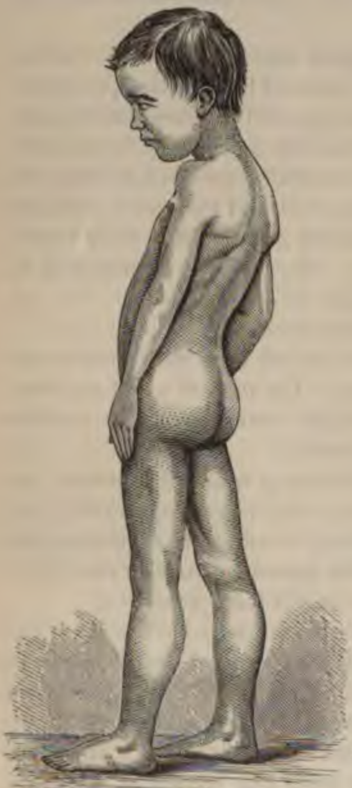
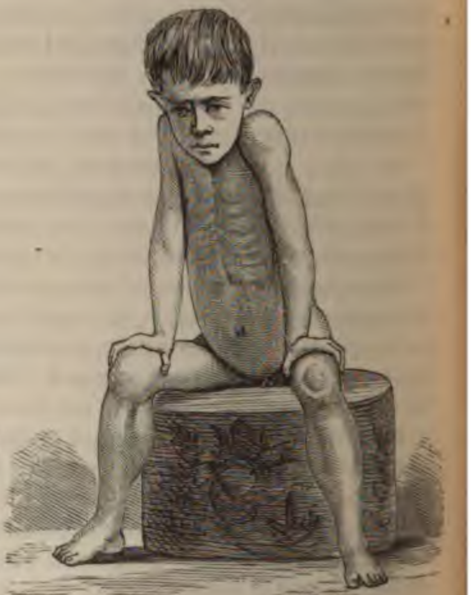


FIG. 54.



The boy's father is a strong and robust man; his mother is delicate, and has had seven children, of whom three are younger than the patient. In two, at least, of the other children there is some enlargement of the gastrocnemii muscles and a general emaciated appearance of the upper extremities. With the birth of the last child the mother had puerperal mania, and, June 23d, had not fully recovered. She had a sister and a brother who were insane, and there is incurable insanity in collateral branches of her family.

Fig. 53, from a photograph, shows well the atrophy of the trunk and upper extremities, the spinal curve, and the hypertrophy of the gastrocnemii muscles. Fig. 54 exhibits the remarkable position assumed by the patient just as he is about to rise from the sitting posture. The atrophy of the muscles of the chest and abdomen is also shown.

Causes.—The disease is one which is almost entirely confined to children, and boys are more liable than girls. Nevertheless, it is not a disease peculiar to very early infancy. Of thirteen cases observed by Duchenne, six are stated to have begun in first infancy, while in seven the inception occurred at from two to ten years. Cases have also been reported as occurring in adults. From a table containing an analysis of forty-one cases given by Dr. Webber, in his paper already cited, it appears that in one case the patient was twenty-six when the disease began, in one a few years under forty, and in one about twenty-eight.

Duchenne expresses the opinion that a hereditary tendency sometimes exists, and this appears to be the fact. Of the cases analyzed by Poore, in two, a maternal uncle and aunt had the disease; in one, three maternal uncles and aunts were affected; in one, one maternal uncle and one half-uncle; in one, three maternal half-brothers; and in one, a maternal half-brother, three maternal uncles, and other members on the mother's side.

The disease does not appear, therefore, to be transmitted directly from parent to offspring, but is a marked example of atavism. The descent is always from the mother's side.

As to exciting causes, little or nothing is known. In none of my cases could any reasonable explanation of its etiology be given. There is some reason for ascribing it occasionally to exposure to cold and dampness, and to antecedent febrile diseases.

Diagnosis.—The only affection at all resembling that under consideration is simple muscular hypertrophy due to an excessive supply of blood being sent to a part of the body. The histories and phenomena of the two disorders are, however, so very different, that I do not see how any error can arise in making a diagnosis between them. Nevertheless, it is tolerably certain that mistakes on this point have been made. Thus, such cases as the one reported by Mr. Maunder,[1] which was clearly one of muscular hypertrophy possessing no analogies with the disease under consideration, have to my knowledge been regarded as instances of the disease under notice.

---

[1] *Medical Times and Gazette*, March 27, 1869.

Duchenne,[1] under this head, gives very elaborate directions for the discrimination of cases of pseudo-hypertrophic paralysis from those of progressive muscular atrophy occurring in infants, infantile paralysis, and the tardy development of the coördinative and motor functions in young children. But it appears to me that very slight inquiry and examination will suffice to make errors in regard to any of these conditions almost impossible on the part of any one capable of distinguishing one disease from another.

Prognosis.—The prognosis is unfavorable. Two cases of recovery are related by Duchenne, and other observers have reported improvements, but the tendency is to death, though life may be prolonged many years notwithstanding the gradual advance of the disease. And yet the fatal result is rarely directly due to the disease itself. Some intercurrent affection ensues, and the vital power being enfeebled cannot resist effectually the new disorder. Thus death occurred in my first case by pneumonia; and of thirteen cases referred to by Poore, in which the termination is given, not one died directly of the disease.

Morbid Anatomy and Pathology.—Five years ago, in the first edition of this work, I wrote as follows: "The spinal cord has only been examined in one case—that of Eulenburg, by Cohnheim—and no lesion was discovered. We are not, from this negative result, to infer that changes had not taken place. About the same time observers were everywhere declaring that in progressive muscular atrophy, organic infantile paralysis, and locomotor ataxia, there were no central lesions. I have no doubt that careful microscopic examination of the spinal cord, after the manner of Dr. Lockhart Clarke, will result in the detection of atrophy and degeneration of nerve-cells in cases of hypertrophy of muscular connective tissue."

Since that time examinations of the spinal cord have been accomplished by several observers, and I think it will be rendered apparent from the following remarks that the prophecy then ventured has been measurably fulfilled and notably by the distinguished physician and histologist to whom reference was specially made. I shall endeavor to place the results of these examinations before the reader, and to state the conclusions which, in my opinion, are to be derived from them.

Eulenburg's case, above referred to, was the first in which the spinal cord was examined; the investigation was conducted by Cohnheim,[2] and the spinal cord was found to be healthy. But as Charcot[3] admits, in bringing forward this case, the method of examination was imperfect and of such a character that "delicate lesions such as atrophy of the motor cells and sclerosis of the anterior horns of gray matter might have escaped observation." We may, therefore, with entire propriety set the results aside, as teaching us nothing definite on the subject.

---

[1] *Op. cit.*, and "De l'électrisation localisée," troisième édition, Paris, 1872, p. 608.

[2] *Verhandlungen der Berliner medicinischen Gesellschaft*, Berlin, 1866, H. ii., p. 191. Also Canstatt's *Jahresbericht*, H. ii., 1866, p. 261.

[3] "Leçons sur les maladies du système nerveux," troisième fascicule, 1874, p. 260.

W. Müller[1] reports the examination of the spinal cord in a case of the disease under notice, which, however, was complicated with profound cerebral disease. He found gray degeneration of the lateral columns and atrophy of the nerve-cells of the anterior horns of gray matter.

Mr. W. B. Kestevan, who had previously reported[2] three so-called cases of pseudo-hypertrophic paralysis associated with imbecility, subsequently examined the brain and spinal cord of one of them who, besides the mental weakness, was subject to epilepsy. The investigation was almost entirely confined to the brain and is reported[3] in such general terms that it is difficult to ascertain to what part of the cerebro-spinal axis the remarks apply. And the plate which accompanies the paper is so imperfectly described that it is impossible to determine what part of the brain or spinal cord it represents, or even to which of these organs it relates.

Nevertheless, it is stated that the perivascular canals were enlarged, that there was granular degeneration, and that the nerve-cells of the brain and spinal cord were normal. On account of the insufficiency of the details, as well as for the reasons that the brain-disease was evidently the primary affection, and that it is exceedingly doubtful from the description given that the case was one of pseudo-hypertrophic paralysis at all, it is not in this connection entitled to much if any weight.

Then Dr. Barth[4] had the opportunity of examining the cord in the case of a man forty-four years old who had suffered from the symptoms of pseudo-hypertrophic paralysis. The loss of power was first manifested in the lower extremities, and was in the early stage accompanied with atrophy as in Dr. Pepper's case. After a time, however, the shrunken muscles began to enlarge, and when the patient died had attained to a great size. Examination made of them established the fact that they exhibited the lesions characteristic of the disease—lesions to which attention will presently be directed.

The lateral columns of the cord were in a state of sclerosis from the upper border of the cervical enlargement to the lower part of the lumbar region. The anterior horns of gray matter were greatly atrophied and many of the nerve-cells had disappeared, while those that remained were shrunken.

---

[1] "Ein Fall vom umschriebener Muskel-Atrophie mit interstitieller Lipomatose," *Beiträge zur pathologischen Anatomie und Physiologie des menschlichen Rückenmarks*, Heft ii., Leipzig. 1870

[2] "Cases of Mental Imbecility associated with 'Duchenne's Paralysis' or Pseudo-hypertrophic Muscular Paralysis," *Journal of Mental Science*, April, 1870, p. 41.

[3] "Microscopical Anatomy of the Brain and Spinal Cord in a Case of Imbecility associated with Duchenne's Paralysis," *Journal of Mental Science*, January, 1871, p. 563.

[4] "Beiträge zur Kenntniss der Atrophia musculorum lipomatosa," *Archiv der Heilkunde*, Leipzig, 1871, p. 120

Still later Charcot[1] minutely examined the cord in the case of a patient of Duchenne's, who had died of an intercurrent affection while the subject of pseudo-hypertrophic paralysis. The results were absolutely negative. The antero-lateral and posterior columns were in a state of perfect integrity, and the gray matter which was made the special object of investigation presented no trace of alteration. The anterior horns were neither atrophied nor deformed, the neuroglia possessed its ordinary transparency, and the motor cells, in number normal, exhibited no deviation from the physiological type.

Last of all, Dr. Lockhart Clarke[2] has given the details of his investigation into the morbid anatomy of the spinal cord in a case of pseudo-hypertrophic paralysis. He ascertained that the spinal cord presented various changes throughout the cervical, dorsal, and lumbar regions. The most important was the disintegration of the gray substance of the anterior, lower, and central portions of each lateral half. In some places this had occurred, chiefly around the vessels, but in others it involved extensive areas ; about the level of the last dorsal nerves it had amounted to almost total destruction of gray matter on each side between the posterior vesicular columns. Other changes, as disintegration of the nerve-roots, commencing sclerosis of the lateral and posterior columns, destruction of the white commissure in various places, dilatation of vessels and extravasations, were noticed.

We therefore perceive that in two cases—Cohnheim's, which, for reasons stated, we may disregard, and Charcot's—there were no lesions found in the cord ; while in three—Müller's, Barth's, and Clarke's—the anterior horns of gray matter were found more or less diseased. Charcot argues very unphilosophically against the legitimate deduction to be drawn from these cases, and, basing his opinion on his own investigations, which led to negative results, contends that the spinal cord is not the seat of the lesion which causes the symptoms of pseudo-hypertrophic paralysis.

But what weight is to be attached to his isolated case when we consider the results obtained by Müller, Barth, and Lockhart Clarke, and compare them with the phenomena of the disease in question, may be determined by reference to a memoir, published in a number of the same journal for the same year that contained his own paper. No one has contended more energetically and with greater success than M. Charcot, for the view that infantile spinal paralysis is a disease, the seat of which is in the anterior horns of gray matter of the spinal cord. Now, the memoir[1] in question details very minutely the state of the muscles of a patient affected from birth with double talipes varus, and it is shown that they were in precisely such a condition as are the affected muscles of a patient the subject of infantile spinal paralysis, and yet the most thorough and careful examination of properly-prepared sections of the spinal cord, taken from the whole lumbo-dorsal region, failed to show the slightest deviation in any respect from the normal standard.

---

[1] "Note sur l'état anatomique des muscles et de la moëlle épinière dans un cas de paralysie pseudo-hypertrophique," *Archives de Physiologie*, Mars, 1872, p. 228.

[2] "On a Case of Pseudo-Hypertrophic Muscular Paralysis," *Medico-Chirurgical Transactions*, vol. xlvii., 1874, p.

In another memoir, on a similar subject,[1] with a like condition of the muscles, one whole lateral half of the cord was atrophied and the nerve-cells of the antero-external group had entirely disappeared.

Doubtless M. Charcot will admit that the positive evidence of M. Dejerine is more convincing in the matter of intra-uterine infantile paralysis than the negative testimony of MM. Coyne and Troisier. In this he would certainly be logical, and I am disposed to accord like precedence to the investigations and affirmative results of Müller, Barth, and Clarke, in comparison with the negative results of M. Charcot's examination.

In Müller's, Barth's, and Clarke's cases, the lesions were not limited to the anterior tract of gray matter, but neither were the phenomena those of simple uncomplicated pseudo-hypertrophic spinal paralysis.

I think, therefore, that, in the present state of our knowledge, we are warranted in at least provisionally accepting the view that the anterior tract of gray matter is the seat of lesion in pseudo-hypertrophic spinal paralysis.

Another very important point in the morbid anatomy of pseudo-hypertrophic spinal paralysis relates to the condition of the affected muscles. Here there is a tolerable uniformity of views.

In the first stage there may be—as Pepper has shown, and as my second case likewise exhibits—atrophy of the muscles instead of hypertrophy. A microscopical examination shows the transverse striæ to be in process of disappearing, and in some of the fibrillæ, to have altogether gone.

The connective tissue already shows a tendency to proliferation, but there is as yet no trace of that fatty degeneration and deposit which afterward becomes the most striking patho-anatomical feature of the disease. In the case which I have detailed, a portion of the primarily-atrophied left rectus femoris muscle was removed by Duchenne's trocar, and, examined with a fourth-inch objective, presented the appearance above described. In Pepper's case not a single fibril of the deltoid muscle which he examined exhibited evidence of fatty degeneration, though the connective tissue was very greatly in excess of the normal proportion, and in places there were small collections of minute fat-globules or refracting granules.

---

[1] "Pied bot varus congenital double. Examen de la moëlle épinière," par MM. Coyne et Troisier, *Archives de Physiologie*, 1871–'72, p. 655.

[2] "Note sur l'état de la moëlle épinière dans un cas de pied bot équin," par J. Dejerine, *Archives de Physiologie*, No. 2, Mars, Avril, 1875, p. 253.

But in the hypertrophied muscles there is not only fatty degeneration of the muscular tissue, but there is fatty deposit between the fibrillæ, and a still more notably-increased development of the connective tissue. As the process advances, the fibrillæ in great part disappear, fat and connective tissue crowding them out as it were, and eventually even this latter is in a great measure replaced by fat-vesicles. The muscle is now at its most advanced stage of hypertrophy. But the process is not yet complete, for a stage of secondary atrophy begins, the fat is absorbed, and finally nothing is left but a few degenerated muscular fibrillæ and a mass of connective tissue.

There is thus in the first place simply a change in the muscular fibrillæ characterized by a disappearance of the transverse striæ. This is probably the first stage of the fatty degeneration, which is afterward manifested unmistakably. At the same time the connective tissue between the bundles of fibrillæ and the fibrillæ themselves, is increased in amount. Then the disintegration of the muscular fibrillæ becomes more evident, the connective tissue is still more increased, and fat-vesicles make their appearance between the fibrillæ and the bundles of fibres. Finally, the muscular tissue mostly disappears, the fat is absorbed, and connective tissue, with perhaps a few fibrillæ, in a more or less advanced stage of degeneration, is all that remains.

In those cases in which there is no primary atrophy of the muscles, the first stage, as described above, is of course wanting.

Relative to the pathology of pseudo-hypertrophic spinal paralysis, there is, as already stated, very decided difference of opinion. Duchenne is undecided. Charcot regards the muscular lesion the essential one—the spinal, when such has been discovered, being accidental. Friedreich[1] is of like opinion as regards the seat of the disease, but considers the affection in question identical in character with progressive muscular atrophy; and Pepper, who, at the date of his writing, had only Cohnheim's imperfectly examined case before him, is apparently disposed to look upon the condition of the muscles as primary; and on the other hand, Müller, Barth, and Lockhart Clarke, consider the spinal lesion the primary, and the muscular the secondary affection. In this view I have already expressed my concurrence.

And I am disposed to agree with the view of Friedreich above referred to, which affirms the similarity existing between pseudo-hypertrophic spinal paralysis and progressive muscular atrophy. This similarity I would not, however, attribute to the mere circumstance that there is an analogy between the states of the muscles in the two diseases, but rather to the fact that the spinal cord is the primary seat of the morbid process in both, and that this process is inflammation of the motor and trophic cells of the anterior tract of gray matter. At the same time, as the diseases are not identical, there is an element of original difference, the nature of which science has not yet been able to unravel.

---

[1] "Ueber progressive Muskelatrophie; ueber wahre und falsche Muskelhypertrophie." Berlin, 1873.

Treatment.—Duchenne, as we have seen, succeeded in curing two cases in their incipiency, with the faradaic current. Authors are agreed that if any thing is likely to prove successful, it is electricity in some one of its forms, and all cases have been treated with this agent. Thus far, however, not only is there no record of another cure, but there is scarcely the mention of even slight improvement. The disease has gone on slowly but certainly in its progress, unchecked by therapeutical measures.

Still we are not, on that account, to despair. I would recommend the primary or galvanic current to the spine, the actual cautery to that part of the spine corresponding to the diseased portion of the cord as determined by the muscular troubles, faradization of the affected muscles with as strong currents as the patient can bear—kneading, the application of heat, and in general those measures which have been recommended for infantile spinal paralysis. Benedict[1] has treated five cases with the primary current to the back, and three of them with the induced current to the affected muscles. These cases were improved. In neither of my cases was there any check to the advance of the disease, though both forms of electricity were assiduously employed.

Internally, strychnia, iron, and phosphorus, may be used, and benefit may be derived from their tonic virtues.

2. *Inflammation of the Motor Cells.*

Thus far, only one disease of this class has been differentiated, and it is characterized by paralysis of the parts involved, without atrophy.

a. *Glosso-Labio-Laryngeal Paralysis.*

The first explicit account of this very remarkable disease is that of Duchenne,[2] who, in consideration of the tendency of the morbid process to advance unchecked, and of the parts affected, designated it "progressive muscular paralysis of the tongue, the veil of the palate, and the lips." The consequences of this condition, as pointed out by Duchenne, are difficulties of articulation and of deglutition, and at a late period of the disease frequent attacks of strangulation, during one of which the patient may die; or death may result either from inanition or syncope.

But, although Duchenne was the first to give a systematic description of the affection, it was observed by Dr. F. W. Robinson, in 1825, who thus writes to Sir Charles Bell:[1] "In consequence of your important discoveries relating to the nerves, I am particularly desirous to have your opinion in the following case: The invalid is an unmarried lady, nearly seventy years of age, who has enjoyed uninterrupted good health up to the present illness. She has had occasional short attacks of gouty rheumatism in both feet and also in the knees, of very short duration. From the first of her complaining up to the present moment, she has been free from headache, and from pain, numbness, or debility of the limbs. The vision and hearing are natural, the appetite good; the bowels regular, and the sleep natural. In short, there is not the slightest deviation from sound health except in the particulars I shall relate.

---

[1] "Electro-therapie," Wien, 1868, p. 186.

[2] "De l'électrisation localisée," etc., deuxième édition, Paris, 1861, p. 621.

"Some few months ago she had some difficulty in using the tongue, and in expressing particular words. This difficulty has gradually increased, and now she cannot protrude the tongue or even move it. She has lost her speech altogether. The tongue itself is soft and pulpy, but it retains its sense of taste and of feeling. The deglutition is impaired, and occasionally she is distressed with a sense of suffocation in attempting to swallow food, which now she is obliged to do with great care. She cannot hack up any thing from the throat nor draw any thing from the posterior nares by a back draught. The features of the face are quite natural, and the skin retains its feeling. The saliva occasionally flows from the mouth."

This is certainly a very accurate description of a case which, although its real nature was not recognized at the time, was undoubtedly an instance of the disease under notice.

Then Trousseau in 1841, just twenty years before the publication of Duchenne's account, recognized it as an affection he had not previously seen, and wrote a memorandum of the existing phenomena.[2] Trousseau named the disease glosso-laryngeal paralysis, in his lecture on the subject, and this was afterward amplified by Duchenne into glosso-labio-laryngeal paralysis. Many cases have been subsequently reported, and descriptions of the affection given, but no one has added any thing to the graphic symptomatology of Duchenne.

Fifteen cases of the disease have come under my observation during the past ten years.

Symptoms.—It rarely happens that patients seek medical advice for the initial symptoms of the disease under notice. We are therefore, in general, obliged to rely on their accounts of the order and progress of the symptoms. In one instance only—and this patient is still under treatment—have I had the opportunity of observing a case from a very early point in the course of the disease.

The first evidence of disease, which in the majority of instances at-

[1] "The Nervous System of the Human Body," London, 1830, p. cxvii.

[2] "Lectures on Clinical Medicine," Bazire's translation, p. 117.

tracts the attention of the patient, is a slight difficulty of articulation, due to a want of rapidity and exactness in the movements of the tongue. This circumstance occurred in eleven of my cases. In the others the symptom first noticed was a tendency in the lips to remain separate, and the consequent necessity of using some degree of mental action to keep them closed. In a short time the restraint in the motions of the tongue becomes more distinctly marked, and it is especially characterized by an inability to raise the extremity to the roof of the mouth, or to press it against the upper teeth. The words, therefore, which the patient experiences most difficulty in pronouncing distinctly are those which begin with lingual or dental consonants. The gutturals he can articulate without trouble; and the labials, except when the affection begins in the lips, do not yet give him inconvenience.

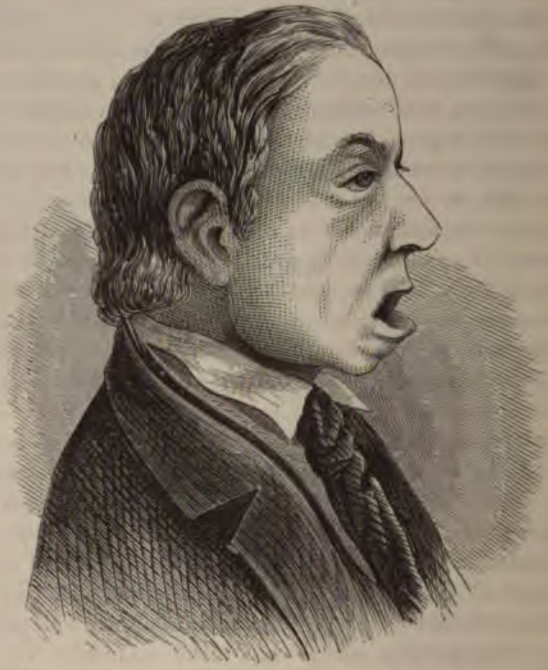
The next symptom to make its appearance is difficulty of swallowing. The food is not promptly grasped by the constrictor muscles of the pharnyx, and the tongue does not press it strongly against them. At times it enters the pharynx, and, not being carried onward by the muscles of deglutition, may slip into the larynx and occasion suffocation. Liquids are especially difficult to swallow, and are often ejected through the nostrils.

As the result of this paralysis of the muscles of deglutition, the saliva, instead of being swallowed as fast as secreted, accumulates in the mouth. Here it becomes stringy from its mixture with the buccal mucus, and when the patient opens his lips it runs out in streams. After a time the orbicularis oris becomes so far paralyzed that the lips cannot be kept closed without continual exertion, and then the viscid saliva is constantly flowing out of the mouth. In four of the cases mentioned as being under my charge, there was from the first some flow of saliva from the mouth, not apparently from any difficulty of swallowing, but from the existing paralysis of the orbicularis oris allowing the mouth to be almost constantly open. The other muscles supplied by the facial nerve in the lower part of the face, singularly enough, do not become involved. The food, it is true, accumulates between the gums and the cheeks, and has to be removed with the finger, but this is not due to any paralysis of the buccinator muscles, but to the want of power in the tongue to move the alimentary bolus around the cavity of the mouth.

When the disease is thus fully developed by the paralysis of the tongue, the veil of the palate, and the lips, the patient presents a pitiable spectacle. He is unable to talk; his teeth are exposed, from the impossibility of closing his mouth; the saliva either runs in streams over the lower lip, or he goes about with a handkerchief in his hand which he uses to absorb the perpetual flow; every attempt at deglutition causes him the utmost distress, and puts him in danger of his life from strangulation. When he opens his mouth the glutinous saliva is seen hanging in viscid strings from the roof, and his tongue, which he cannot move, lies torpid, like an inert mass of muscles as it is.

The facial expression is well seen in the accompanying woodcut (Fig. 55), made from a very accurate sketch of one of my patients suffering from the disease in question, and who entered my consulting-room with his handkerchief to his mouth to absorb the streams of saliva which were flowing.

Fig. 55.



The condition of the patient becomes still more painful from the implication of the respiratory muscles. The walls of the chest become paralyzed, and he is unable not only to breathe deeply, but to cough so as to keep the bronchial tubes clear of accumulations of mucus. So feeble is the respiratory power, that with all the effort he can make he cannot blow out a candle.

And, besides the impossibility of articulation, the larynx becomes paralyzed at a later period of the disease, and phonation becomes impossible. The patient is then doomed to perpetual silence, even the power of whispering being lost.

A remarkable fact is characteristic of many cases of glosso-labio-laryngeal paralysis, and that is the tendency of the morbid action to extend so as to implicate other nerve-cells lower down in the spinal cord. But the cells thus affected are not motor, but trophic, and as a consequence the resulting condition is not paralysis but muscular atrophy. In none of my cases was there muscular atrophy in any part of the body, but in one, to be presently referred to more at length, there was incipient paralysis of the right arm. The case was, therefore, similar to the one reported by MM. Duchenne and Joffroy, and which will be more specifically referred to hereafter.

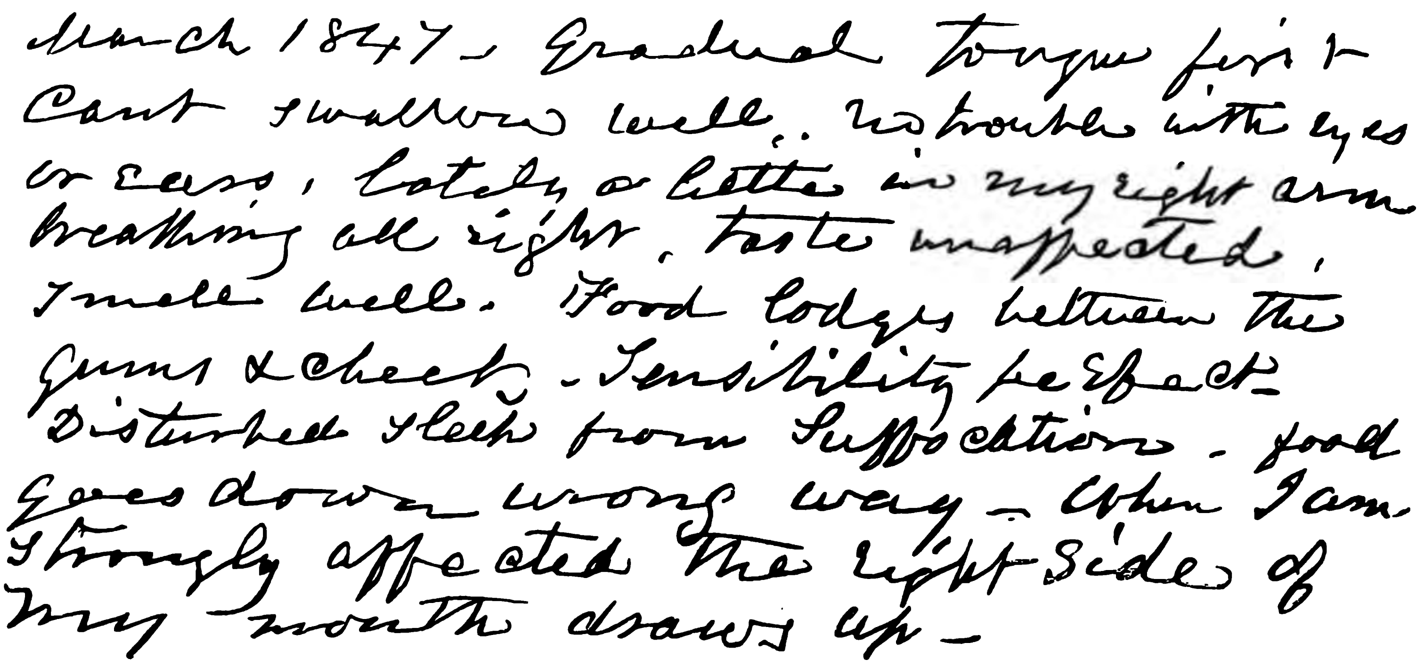
Gradually, as the disease advances, the physical powers of the patient yield. He becomes unable to walk, not from paralysis, but from general debility, due to insufficient nutrition and imperfect respiration. His appetite remains good, but he is afraid to take any more food than is barely sufficient to sustain life, for experience has taught him that suffering and danger are attendant on every attempt at deglutition. At last he ceases to make the effort, and is fed with liquid food through a stomach-tube. The saliva during sleep runs down his throat, and fits of suffocation are the result. Too weak to walk, he remains in bed, his head turned to one side so as to allow free egress for the saliva, and he dies either from asphyxia, from the cessation of the action of the heart through the continued extension of the lesion to the cells supplying the pneumogastric nerve, or from some intercurrent affection.

Generally the mind remains clear to the last, but in a very interesting instance of the disease occurring in an officer of the army, sent to me by my friend Dr. Fleming, of Pittsburg, this was not the case, manifest dementia making its appearance toward the close. The emotions are, however, almost invariably easily excited.

The first case of this disease coming under my observation was one referred to me, over eight years ago, by my friend Dr. Edward Bradley, of this city. The patient was a watchmaker, and very intelligent. Though unable to speak a word, I obtained a good deal of information from him relative to his disease by asking him questions, the answers to which he wrote. The accompanying *fac-simile* of one of his written communications to me (Fig. 56) will, I doubt not, prove of interest. It was made partially in answer to questions, and partially at his own suggestion. The date (March, 1847) was given in answer to my question when the disease appeared, and the year mentioned is a mistake for 1867. As he states, there was a little trouble with his right arm. This was of the nature of paralysis, there being no muscular atrophy anywhere. The patient died about six months after I saw him, the disease lasting a little over a year.

Another case—the eighth—was a patient in the New York State Hospital for Diseases of the Nervous System. In him the affection began in the orbicularis oris, and gradually involved the tongue and muscles of deglutition. The left side was first affected, and then, a few weeks afterward, the paralysis extended to the right. There was nystagmus of both eyes. The mind was perfectly clear. He formed

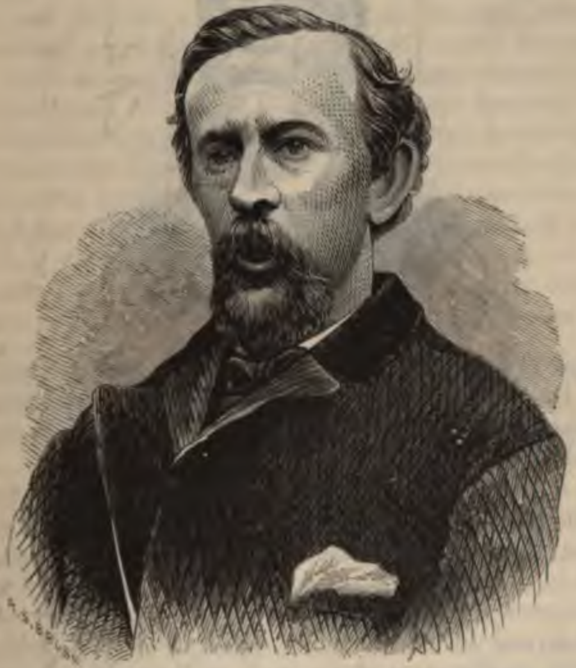
Fig. 56.



March 1847 - Gradual tongue first
Cant swallow well.. no trouble with eyes
or ears, lately a little in my right arm
breathing all right, taste unaffected,
Smell well. Food lodges between the
gums & cheek - Sensibility perfect -
Disturbed sleep from Suffocation - food
Goes down wrong way - When I am
Strongly affected the right Side of
my mouth draws up -

e subject of a clinical lecture on glosso-labio-laryngeal paralysis, iich I delivered during the session of 1870–'71, at the Bellevue Hos-al Medical College. The case is further remarkable as occurring in exceptionally young person, the patient being but thirty-two years age. Duchenne[1] states that he has never observed it in persons der forty. I subjoin a representation of this patient (Fig. 57), taken

Fig. 57.



m a photograph. The paralysis of the orbicularis oris is evident, al-ough it is partly concealed by the mustache. At the time it was ken the patient could swallow, but was conscious of a difficulty in ginning the act of deglutition.

In this case the first symptom observed by the patient was a marked næsthesia of the face and lining membrane of the cheek on the left ide. Krishaber[2] has since reported an instance of like character, and egards the loss of sensibility as a valuable precursory sign, and as ex-ibiting in a very striking manner the physiognomy of the disease.

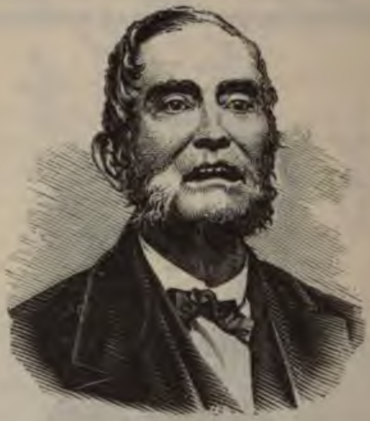
I subjoin the engraving (Fig. 58), from a photograph, representing a atient who came from the West to consult Dr. Sayre and myself. He entered my consulting-room holding his handkerchief to his mouth, to catch the streams of saliva which were pouring from it, unable to speak a word and scarcely able to swallow.

---

[1] "De l'électrisation localisée," Paris, 1861, p. 648.

[2] "Anæsthesie de la sensibilité réflex des voies aériennes et digestives, comme pré-rseur de la paralysie labio-glasso-laryngée," *Gazette hebdomadaire*, November 29, 1872.

FIG. 58.



Causes.—The etiology of glosso-labio-laryngeal paralysis is very obscure. Duchenne attributes one of his cases to mental anxiety; two cases appeared to be due to syphilis and rheumatism. In no other instance could he assign a cause.

Of my own cases, one was apparently due to business troubles resulting from petroleum speculations; and, in one, excessive application to business appeared to be the cause. In one other case, that of a gentleman of this city, the disease was evidently associated with syphilis, and in one it was apparently caused by a blow on the back of the head, and in one by exposure to a strong draught of cold air, which blew directly on the nape of the neck and occiput. In none of the others could I assign any cause. All of my patients were between the ages of forty and sixty, except the one whose case and portrait (Fig. 57) have been given.

Diagnosis.—Attention to the account of the symptoms given will prevent any mistake in diagnosis, as there is no affection which resembles in its entirety the one under consideration. In the very early stage, however, it may be confounded with simple paralysis of the tongue; or, if the disease begins in the lips, as in the case cited, with facial paralysis. In glossoplegia there are other symptoms of cerebral disorder, and in facial paralysis the loss of power is not confined to the lips.

It may possibly, in some cases, not be distinguished from the general paralysis of the insane, which generally begins with paralysis of the tongue and weakness of the lips. The facts that this disease is manifested also by mental symptoms, and that the paralysis gradually involves the other muscles of the body, will suffice for making an exact diagnosis. In facial diplegia the expression of countenance is very much like that of a patient suffering from glosso-labio-laryngeal paralysis, but here the resemblance ends, and careful examination shows even here many points of difference. It is only necessary to state that the tongue is not paralyzed, and that there is no difficulty of swallowing in double facial paralysis.

In progressive muscular atrophy, attacking the tongue, the veil of the palate, and the lips, a mistake might also be made. But, as Duchenne remarks, progressive muscular atrophy rarely begins in that way, and, when it does, other muscles of the body, especially the thenar and hypothenar eminences, will soon become involved. Charcot has, however, recently reported a case, to be presently more fully quoted, in which progressive muscular atrophy was clearly combined with glosso-labio-laryngeal paralysis, and in which, on post-mortem examination, though the volume of the tongue was not diminished, the muscular fibre had undergone degradation. In such a case, of course, a complete diagnosis could only be made after death. In ordinary progressive muscular atrophy, the fact that the atrophy comes on before the paralysis, is to be borne in mind.

From diphtheritic paralysis attacking the muscles of the pharynx, glosso-labio-laryngeal paralysis is readily distinguished by inquiries relative to the history of the case, and by the fact that the tongue is not involved in the first-named disorder.

Prognosis.—There is no instance on record of a cure.

All my patients affected with the disease are dead, except one whom I occasionally see. A case of improvement and one of cure have been reported by Dr. Cheadle,[1] but certainly neither was an instance of glosso-labio-laryngeal paralysis, although the face, the tongue, and muscles of deglutition, may have been paralyzed. In the first of these the disease began with sudden loss of speech, then retroceded, then returned. There was facial paralysis, incontinence of urine, and left hemiplegia. The woodcut, from a photograph, of this case does not exhibit a single feature of glosso-labio-laryngeal paralysis. The case was probably one of syphilitic basilar meningitis, and the patient greatly improved "under iodide of potassium, rest, and nutritious food," and was discharged able to swallow with very little difficulty and to articulate imperfectly, indeed, but so as to be understood.

In the second case a complete cure was effected, and, as indicative of the character of the disease, I subjoin the essential parts of Dr. Cheadle's report :

A woman, aged forty-two, entered St. Mary's Hospital in November, 1867. Her speech was so much affected that it was difficult to make out a word of what she attempted to say; but, from the statement of a fellow-servant who accompanied her, and her own subsequent statements, the following history was elicited: For some months she had suffered from frequent attacks of violent shooting pain in the head, accompanied by dimness of vision, and quite unlike any headache she ever felt before. With this exception, she had remained in good health till a few days before she applied for medical aid, when she was suddenly seized, while sitting in a chair in the daytime, with total loss of speech and paralysis of the right side. Her face was drawn, the right arm and leg utterly useless, and she found herself only able to utter inarticulate sounds; there was no loss of consciousness, or it was so transitory as to escape observation. The use of the leg was fully regained in about a week; but the arm remained weak for a considerable time. For two days speech was so far abolished that she could only utter inarticulate sounds.

---

[1] "Labio-Glosso-Laryngeal Paralysis," "St. George's Hospital Reports," vol. v., 1871, p. 123.

When fully examined several weeks after the attack, it was found that she could walk perfectly well, but that the arm was weak and sensation slightly impaired. Her speech was thick, indistinct, and nasal, and she was not able to protrude the tongue fully. The condition of the lingual, palatal, and facial nerves was not accurately ascertained. She complained of severe shooting pains in the head, and of extreme drowsiness.

She had had four still-born children; and an eruption, which she said was very much like small-pox, made its appearance shortly after her first confinement.

She took small doses of iodide of potassium, but there was no improvement. Mercury was also given, without good result. She then came to the hospital. Articulation was still very indistinct; she spoke as one very drunk, and was quite unintelligible. In reply to questions addressed to her, she had uttered meaningless sounds.

The treatment was continued, and she gradually improved, so that at last she spoke with perfect fluency and clear articulation.

No one, who has ever seen and studied a single case of glosso-labio-laryngeal paralysis, could mistake this case of Dr. Cheadle's for one of the disease described by Duchenne. It was probably a case of syphilitic cerebral disease like the first, and like it recovery took place under anti-syphilitic treatment. Ameliorations may certainly be produced, but probably no cure. The average duration of the disease is about two years.

Morbid Anatomy and Pathology.—Previous to the very recent researches which have given us a clear insight into the morbid anatomy of glosso-labio-laryngeal paralysis, the lesions, detected by several observers, were atrophy of the roots of the hypoglossal, facial, spinal accessory, and pneumogastric nerves. But late investigations have shown that the lesions of the nerve-roots are secondary to others more central in their situation.

It has already been shown, in this chapter, that the morbid process in certain diseases consists of atrophy and disappearance of nerve-cells forming the nuclei of origin of certain nerves. Very minute examinations, made in the cases of persons dying of the disease under notice, show very clearly that it also consists of atrophy and disappearance of nerve-cells.

Thus, in the case cited from Charcot,[1] the tongue had preserved its former thickness and normal dimensions, but the patient could not articulate, and was obliged to express herself by signs. Intelligence was perfect. There was some atrophy of the arms.

The post-mortem examination showed that the extrinsic muscles of the tongue, and those of the supra- and sub-hyoidean regions, were of normal appearance and condition. The intrinsic muscles were pale and of diminished hardness.

The laryngeal muscles were healthy, except the posterior crico-arytenoid and crico-thyroideus, and presented here and there a yellow coloration.

The muscles of the pharynx had undergone no appreciable alteration. The muscular coat of the œsophagus appeared to be of normal consistence and color.

In the spinal cord the alterations were confined to the anterior horns of gray matter, and to the proper nerve-elements, the neuroglia being healthy. The abnormal condition consisted in a disappearance of nerve-cells.

In the bulbar region it was noticed that the nucleus of the hypoglossal presented very pronounced alterations, which here, as below, related exclusively to the nerve-cells. The neuroglia was intact. Many of the cells were in a state of pigmentary degeneration. The group of cells, considered by Lockhart Clarke to be the inferior nucleus of the facial, were smaller, and less in number than in the normal state. The other cells constituting the nucleus of the facial were in like condition. Similar changes were observed in the cells in relation with the filaments of origin of the spinal accessory and the pneumogastric nerves.

In the case which Duchenne[2] has made the basis of some original views on the subject of atrophy of nerve-cells, and to which reference has already been made, it was found that the cells constituting the nuclei of origin of the hypoglossal, the facial, the spinal accessory, and the pneumogastric, had become—those that remained—affected with pigmentary degeneration, and were atrophied, while many had disappeared altogether.

---

[1] "Note sur un cas de paralysie glosso-laryngée suivi d'autopsie," *Archives de physiologie*, tome iii., 1870, p. 247.

[2] "De l'atrophie aiguë et chronique des cellules nerveuses de la moëlle et du bulbe rachidienne, à propos d'un observation de paralysie glosso-labio-laryngée," par Duchenne (de Boulogne) et Joffroy, *Archives de physiologie*, No. 4, 1870.

Among the earliest properly conducted examinations of the medulla oblongata is that made by Dr. E. R. Hun,[1] of Albany, in a case which appears to have been a typical one of glosso-labio-laryngeal paralysis. Sections made from the medulla oblongata showed disappearance of the nerve-cells and hyperplasia of the neuroglia in that part where were situated the nuclei of origin of the facial and hypoglossal nerves. The cells that remained had, in many cases, lost their radiating processes, and were in a state of pigmentary degeneration.

In this case there were in addition symptoms indicating the existence of secondary amyotrophic lateral spinal sclerosis, as described by Bouchard and Charcot, and the lateral columns of the cord were found sclerosed.

It may, therefore, be considered as satisfactorily determined that the essential lesion in glosso-labio-laryngeal paralysis is found in the medulla oblongata and upper part of the spinal cord, and that it consists of atrophy and disappearance of certain nerve-cells constituting the nuclei of origin of the hypoglossal, the facial, the spinal accessory, and the pneumogastric nerves.

But we are not on that account to disregard the fact that phenomena similar to those of glosso-labio-laryngeal paralysis may exist, and as the result of very different lesions of the medulla oblongata, or even of no discernible morbid condition of that organ. Thus in a case reported by Dumesnil[2]—in which there was paralysis of the tongue, the lips, and the veil of the palate, together with atrophy of the muscles of one of the upper extremities—the hypoglossal, facial, and spinal accessory nerves were found atrophied. No thorough microscopical examination was made of the medulla oblongata, and hence such lesions as those described by Charcot and Duchenne were not detected. But, whether they were present or not, it is undoubtedly true that eccentric lesions of these nerves would cause paralysis of the parts involved in glosso-labio-laryngeal paralysis.

Trousseau[3] has described three cases in which post-mortem examinations were made. In one of these, the results were negative from incompleteness of the investigation; in the second, the roots of the hypoglossal nerve were atrophied, and the medulla oblongata was harder than was normal; and, in the third, the roots of the hypoglossal and spinal accessory were in like condition.

In all of these cases no proper microscopical examination was made of the medulla oblongata, and consequently we are without information as to the exact condition of that organ. But we can remark of these ses, as of Dumesnil's, that they only show that paralysis may be oduced by lesions of the nerves, a fact which required no further monstration than it had already received many centuries ago. It arcely, however, admits of a doubt that the atrophy of the nerves as the result of central disease, and that this disease was situated in e medulla oblongata.

---

[1] "Labio-Glosso-Laryngeal Paralysis," *American Journal of Insanity*, 1871, p. 194.

[2] *Gazette hebdomadaire*, Juin, 1859, p. 390.

[3] "Lectures on Clinical Medicine," Bazire's translation, 1866, p. 117, *et seq.*

In Dr. Wilks's[1] case, the roots of the hypoglossal and spinal accesry nerves were found atrophied, and the medulla oblongata was evintly the seat of serious disease, but no examination as to the cellsions was made, nor indeed was it possible then, before the researches Lockhart Clarke, to make such an examination.

Voisin[2] reports the case of a patient aged seventy-seven, who enred the Salpêtrière, and who soon after admission suddenly lost her eech. Gradually, however, she reacquired the power, though she id forgotten some words. After remaining three months in the hostal, she again, after a violent fit of excitement, was deprived of eech, and also lost the power to purse up the lips and to raise the ngue. The mastication and deglutition of solid substances were possible, the saliva flowed from the mouth, the uvula was immovale, the inspiration rattling, and the respiration generally difficult. Taste ad sight extinguished. The glottis was unfortunately not examined. he mind was unimpaired, and there was no paralysis of the limbs. he patient had to be nourished through an œsophageal tube. She ied suddenly after the last attack.

Now, although this is called by Voisin a case of glosso-pharyngoabial paralysis, a title which he uses as synonymous with glosso-labioaryngeal paralysis, it is very evidently not the affection originating in the nuclei of the bulbar nerves, and progressing slowly but without intermission to a fatal termination. It is of the same character as the cases cited from Dr. Cheadle, and would not be referred to here but for the fact that a post-mortem examination was made. The results were as follows :

There was a small yellow focus of softening at the external part of the left lenticular ganglion, which extended to the island of Reil. To this circumstance the reporter attributes the amnesic aphasia.

At the upper and lower surfaces of the two lesser cerebral hemispheres, just beneath the connecting arm of each, were discovered two umors which appeared to be epithelioma of the arachnoid. The left umor, of the size of a walnut, reached to the medulla oblongata, in such manner that the auditory, facial, hypoglossal, and spinal accessory and glosso-pharyngeal nerves were compressed. These nerves were by ne-half slenderer than those of the right side. The facial was softened. The tumor on the right side was of smaller circumference, and did not extend to the medulla oblongata.

---

[1] "Guy's Hospital Reports," vol. xv., p. 1.

[2] *Annales médico-psychologiques*, January, 1871, analyzed in the *Journal of Psychogical Medicine*, New York, vol. v., 1871, p. 816.

Neither the medulla oblongata nor the pons was sclerotic.

No microscopic examination of the medulla was made, and therefore nothing can be inferred relative to the state of the nerve nuclei.

In a case in which I had the opportunity of making a post-mortem examination, there was also paralysis of the tongue, the lips, and the pharynx, but the associated phenomena were not such as to warrant the disease being designated as an inflammation of the anterior tract of gray matter, causing glosso-labio-laryngeal paralysis. The patient was an elderly gentleman of this city, who had suffered from paralysis of the lower extremities, and to a less extent of the arms, for several years. This condition had been preceded by several seizures not involving loss of consciousness, but mainly characterized by deprivation of speech, irregular respiration and circulation, and vomiting.

When I first saw him there was defective articulation, the tongue could only be slightly moved, and there was partial paralysis of both sides of the face. The function of deglutition was very much impaired. Solids could not be swallowed at all, and liquids escaped through the nostrils. The saliva ran in streams from the mouth.

But the most marked disturbance was in the respiration and the action of the heart, both of which were exceedingly irregular, the latter intermitting frequently, and generally not skipping a single beat, but two or three at once. His mind was unimpaired.

I predicted his death in a few days, for, from the history of the case as well as from the existing phenomena, I was convinced that the nucleus of the pneumogastric was involved with that of the hypoglossal, facial, and spinal accessory of both sides, and that the disease was advancing. He died within a week, and a post-mortem examination was allowed.

The brain was apparently healthy throughout, except that the pons Varolii and medulla oblongata were in a state of extreme softening. These were removed, together with the vertebral arteries as far down as the lower border of the anterior pyramids, and with the basilar and its transverse branches. The coats of the basilar were thickened, and the lumen of the vessels almost entirely obliterated. The two lower transverse branches on either side were entirely closed by dense fibrous clots, presenting all the appearance of thrombi. The left vertebral artery was also diseased and closed by an old clot extending about an inch and a quarter from its junction with the vertebral of the opposite side. The tissue of the pons Varolii and medulla oblongata was so much softened as not to admit of hardening in chromic acid. The parts were placed in absolute alcohol and examined in a few days, but the degeneration was so thorough that nothing more could be ascertained than the fact of the almost complete destruction of the nerve-elements.

In this case, although the symptoms were in some respects similar to those of glosso-labio-laryngeal paralysis, yet it is very obvious that the affection was not this disease. The paralysis of the extremities and the paroxysms of speechlessness were indicative of a more extensive and different lesion, and the post-mortem examination showed that the original trouble was altogether extrinsic. The bulb was invaded from the exterior instead of from the interior.

It would be just as proper to designate the case described on page 486, under the head of spinal paralysis of adults, one of glosso-labio-laryngeal paralysis on account of the bulbar symptoms, as to consider the one just described and others of its class as coming under this category. That ischæmia of the medulla oblongata, however, will give rise to the symptoms of glosso-labio-laryngeal paralysis is not only evident from pathological considerations, but also from recent anatomical researches. Thus Duret[1] concludes, as the results of his investigations, that, when a clot is situated in one of the vertebral arteries, it interrupts the circulation in the anterior spinal artery, and consequently in the median arteries which arise from it; that is to say in the arteries which supply the nuclei of the spinal accessory, the hypoglossal and the inferior root of the facial. It therefore causes the development of the symptoms of glosso-labio-laryngeal paralysis. When the clot occupies the inferior part of the basilar trunk, it cuts off the blood from the subprotuberantial branches which supply the nucleus of the pneumogastric, and sudden or at least rapid death is the consequence.

In regard to the character of the morbid process by which the degeneration, atrophy, and disappearance of the nerve-cells are effected, Leyden[2] considers it to be a myelitis, and this is probably the correct view. In this light, therefore, it does not differ essentially from the corresponding process which, situated lower down in the cord, produces infantile spinal paralysis and the spinal paralysis of adults.

Wachsmuth,[3] who was among the first to study the subject, argued, from a consideration of the symptoms, that the affection in question was characterized by destruction of the nerve-cells in the floor of the fourth ventricle, and that the degeneration of the nerve-roots was a secondary process. As we have seen, it was reserved for Charcot and Duchenne and Joffroy to establish the correctness of this opinion by post-mortem investigations.

As to the acute glosso-labio-laryngeal paralysis, or acute bulbar paralysis, as it has been called by Leyden[4] and other German writers, and the "glosso-labio-laryngeal paralysis of apoplectic form" of Joffroy[1] and of Proust,[2] cases such as those described by these authors, are to be considered in the light of the foregoing remarks, and not as instances of inflammation of the anterior tract of gray matter leading to destruction of the motor cells.

---

[1] "Sur la distribution des artères nouricières du bulbe rachidien," *Archives de physiologie*, 1873, p. 97.

[2] "Ueber progressive Bulbärparalyse," *Archiv für Psychiatrie und Nervenkrankheiten*, B. ii. und iii., 1870–'73.

[3] "Ueber progressive Bulbärparalyse und diplegia facialis," Dorpat, 1861.

[4] "Klinik der Nervenkrankheiten," Berlin, 1875, B. ii., p. 157.

In regard to the coexistence ef glosso-labio-laryngeal paralysis with certain affections of the cord, characterized by atrophy of the muscles, the point will be fully considered under the heads of progressive muscular atrophy and amyotrophic lateral spinal sclerosis.

One other point: What is the essential physiological character of the cells which have become degenerated, atrophied, and many of which have disappeared? In infantile spinal paralysis, and in the spinal paralysis of adults, we have seen reason to think that the cells which are the seat of disease are both motor and trophic, for these affections are evidenced by paralysis and atrophy. But in the disease under consideration there is no atrophy, for glosso-labio-laryngeal paralysis is not a disease in which the muscles are defectively nourished, but one the essential feature of which is paralysis. It is reasonable, therefore, to suppose, with Duchenne, that the nerve-cells which have become diseased are motor cells.

Onimus[3] asserts that there is no evidence to show that glosso-labio-laryngeal paralysis ever exists without atrophy of the tongue, but this is directly at variance with the experience of other observers and altogether inconsistent with my own investigations. That there is a form of progressive atrophy affecting the tongue is very certain, but it is not glosso-labio-laryngeal paralysis. As regards the relation of the symptoms observed to the known distribution and functions of the nerves concerned, there is no difficulty. The affection of the hypoglossal causes the paralysis of the tongue, and the consequent impossibility of articulation, and of moving the food in the mouth; the implication of the facial accounts for the paralysis of the lips and the muscles of the veil of the palate, and the resultant impossibility of sounding certain letters, and of swallowing; the extension to the spinal accessory explains the paralysis of the larynx, the loss of phonation, and the feebleness of respiration; and death, when it takes place as it sometimes does from the sudden stoppage of the heart's action, is due to the implication of the pneumogastric, to which cause other paralyses of the muscles of animal life are to be ascribed.

Treatment.—From what was said relative to the prognosis, it will have been perceived that there is not much to expect from treatment. I have, however, occasionally produced good results which have, for a time at least, rendered the condition of the patient more tolerable. Thus, the first patient who came under my care was much relieved by faradization of the paralyzed muscles. He improved very much in his ability to swallow, and in power over his tongue and lips. These ameliorations were not permanent. In the case of the gentleman from Pittsburg, as well as in all the other cases but one, similar treatment, together with the use of the primary galvanic current and phosphorus, was without the least effect. In this latter case some benefit was apparently produced for a time. The course of the disease was certainly less rapid than before treatment was begun, but it nevertheless slowly advanced to a fatal termination.

---

[1] "Sur un cas de paralysie labio-glosso-laryngée à forme apoplectique d'origine bulbaire," *Gazette Médicale*, 1872, No. 41.

[2] "Sur la paralysie labio-glosso-laryngée," *Gazette des Hôpitaux*, 1870.

[3] "Paralysie labio-glosso-laryngée," *Gazette des Hôpitaux*, September 30, 1872.

3. *Inflammation of the Trophic Cells.*

In admitting the existence of trophic cells in the anterior tract of gray matter of the spinal cord, I have been influenced by what I consider to be the weight of evidence. The fact is one which, in the present state of our knowledge, is not capable of absolute demonstration; but the subject is of such a character as scarcely to require proof of that nature. The inference for their existence is as strong as that which we draw relative to the presence in the spinal cord of cells specially in relation with the functions of motion and of sensation. As we have seen, there are affections of the cord in which there are both paralysis and atrophy. In such cases, we have good reason for concluding that the cells which are in nervous connection with the paralyzed and atrophied muscles have both motor and nutrient properties. This deduction is strengthened by the fact that there is another disease which is characterized by the existence of paralysis without atrophy, and which post-mortem examination shows to be due to the degeneration, under the influence of inflammation, of certain cells situated in the medulla oblongata and in direct anatomical relation with the nerves supplying the paralyzed parts. These cells, there is every reason to believe, are exclusively motor.

We have now to consider the affections of the spinal cord, and still of the anterior tract of gray matter, which are manifested by atrophy without paralysis, except in so far as an atrophied muscle necessarily possesses less power than one which is of full size.

Two such affections have been recognized, or, rather, one—progressive muscular atrophy—has been regarded as a disease of the anterior tract of gray matter of the spinal cord by the great weight of authority; while the other—facial atrophy—is now for the first time, so far as I am aware, placed in the same category. That this is warranted by the clinical histories of the cases I shall have to adduce, will, I think, be apparent to the reader.

a. Progressive Muscular Atrophy.

Although cases of progressive muscular atrophy were noticed by the older writers, the first systematic account of the disease was given by Duchenne,[1] in 1849. In 1850 M. Aran[2] published his memoir, in which he gives the histories of eleven cases; and three years subsequently Cruveilhier[3] read a paper on the same subject before the Académie de Médecine. About the same time other memoirs were published on the subject.

But, although Cruveilhier was not the first to write upon the affection in question, he was the first to describe it, and Duchenne and Aran were aware that he had done so in his lectures for several years. The disease is therefore sometimes called Cruveilhier's atrophy.

Symptoms.—The first symptom observed in the majority of cases is loss of strength and dexterity in certain muscles of the body. If these are in the lower extremities, the patient finds that he tires in walking sooner than he used to do. If in the upper extremities, he experiences weakness in the shoulder, arm, or hand, according to the muscles affected.

Soon afterward pains simulating those of neuralgia are felt in the paretic muscles. These are not probably due to the central lesion, but are the result of muscular fatigue which is itself due to the incipient atrophy which even at this stage exists.

In the majority of cases—according to my experience in all—fibrillary contractions are perceived. Thus, of fifty-two cases of progressive muscular atrophy which have been under my charge during the past ten years, these contractions formed a prominent feature in every one. They consist of slight twitchings of separate bundles of muscular fibres, and give the sensation of something alive being under the skin. They can often be seen, especially when superficial fibres are involved, and they are generally the *avant courriers* indicating the extension of the disorder. Even if for a time they are not noticed they can always be excited by a smart tap on the atrophied muscle, except in the latter stages of the disease.

The loss of strength attracts the attention of the patient to his limbs, and then he finds that the weakness is accompanied by atrophy. If, as is usually the case, the disease begins in one of the upper extremities, the thenar and hypothenar eminences very commonly give the first evidence of atrophy. The ball of the thumb disappears, and the muscles filling the space between the first and second metacarpal bones

---

[1] "Atrophie musculaire avec transformation graisseuse," "Mémoires de l'académie des sciences," 1849.

[2] "Recherches sur une maladie non encore décrite du système musculaire," *Archives Générale de Médecine*, 1850.

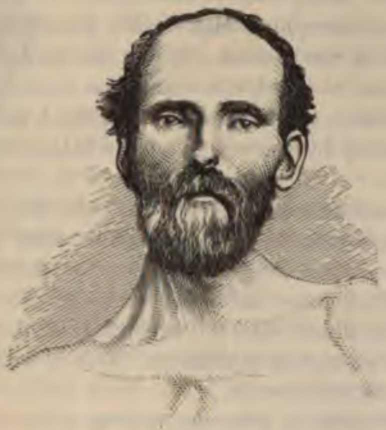
[3] "Sur la paralysie musculaire progressive atrophique," *Archives Générale de Médecine*, 1853.

—the adductor pollicis and the first interosseous—likewise shrink away. The whole outline of the metacarpal bone of the thumb can thus, very soon, easily be made out.

The ball of the thumb is often the starting-point of the disease, and, when this is not the case, it generally becomes involved at some time or other in the course of the affection. Of the fifty-two cases occurring in my experience, the disease appeared first in the ball of the thumb in nineteen, and eventually attacked this part in twenty-one others. The upper extremities were the original seat of the disease in forty-two cases, the trunk in four, and the lower extremities in six. Whether the affection begins in an upper or lower extremity, the tendency is for the opposite member to be next involved.

The physiognomy of progressive muscular atrophy is very striking, particularly when the face or the hand is its seat. One very well-marked case of the former has come under my observation, and it can readily be understood that the change effected by the disappearance of the facial muscles must be very evident. In the case in question—represented in Fig. 59—the right side of the face was strikingly involved, and the muscles of the neck and shoulder on the same side were affected to a marked degree. In the hand, the atrophy of the muscles which give this member its plumpness, and enable it to perform the complex movements of which the fingers are capable, causes appearances which are easily recognizable. By the disappearance of the thenar eminence, the skin over it hangs in loose folds, the thumb falls by its own weight, and cannot be brought into apposition with the index-finger—the palm of the hand is hollowed out, and the metacarpal bones can be distinctly seen and felt.

FIG. 59.



In the forearm, the situation of the disease can be readily ascertained by the flattening produced by the disappearance of the affected muscles, and in the arm and shoulder the effects of the disease are still more evident. In three cases, the disease had begun in the right deltoid, and had not extended beyond this muscle when the patients came under my charge. In all, the shoulder was flattened, and the head of the humerus and the acromion process could be distinctly seen. In another case it was limited to the trapezius and scapular muscles of both sides.

In the lower extremity, the changes in the foot are not so remarkable as the corresponding ones in the hand, but the effects produced by the atrophy of the peroneal muscles, the tibialis anticus, and those forming the calf of the leg, are very striking. In the one case, the foot drops, and the patient is obliged to bend the knee to a greater extent than usual in order to make the toes clear the ground; in the other, the heel cannot be raised, and the ankle gives way with the weight of the body. When the muscles on the anterior face of the leg are in process of destruction, the forms of the tibia and fibula can be distinguished, and the space between the two bones is unfilled. The disappearance of the calf makes the posterior aspect of the leg flat.

In the thighs the atrophy is also readily perceived, and modifies very materially the gait of the patient. When the extensors on the anterior face of the thigh are involved, the leg cannot be thrown forward; when the flexors are the seat, the leg cannot be raised, and the whole member has to be lifted up by the action of the flexors of the thigh on the pelvis.

A singular circumstance connected with the disease is the tendency exhibited for a single muscle or a group of muscles to escape atrophy, while all the surrounding ones are profoundly affected. Thus, as in a case reported by Duchenne,[1] all the muscles of the hand and forearm were completely atrophied with the exception of the supinator longus, which remained in its normal condition. This is well shown in the cut (Fig. 60) from Duchenne's work.

Sometimes the atrophy, after destroying a muscle or two, ceases to extend. Thus, in a case referred to me by Dr. D. H. Goodwillie, of this city, the atrophic process had been spontaneously arrested after completely destroying the muscles of the right thenar eminence, and the patient had remained for eighteen years entirely free from any active manifestations of the disease.

The temperature of the atrophied muscles is usually several degrees below the normal standard. In the case of a gentleman whom I recently examined with reference to this point, and whose right hand, forearm, and arm, are in a state of advanced atrophy, I found, by means of nbard's instrument, the temperature of that extremity to be 5° elow that of the other.

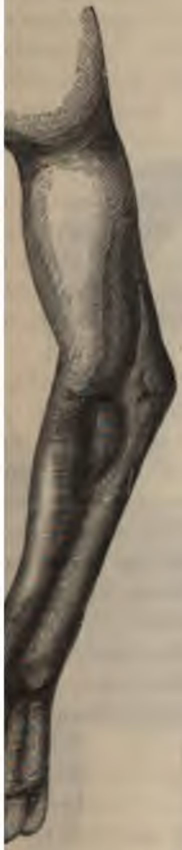
---

[1] "De l'électrisation localisée," troisième édition, Paris, 1872, p. 506.

cutaneous capillaries are usually relaxed, and hence the skin e affected parts is discolored by the passive engorgement.

electric contractility of the affected muscles is not diminished so there are any muscular fibres to be acted upon. As the morbid process generally pursues no regular course in its progress through a muscle, but takes bundles of fibres here and there without regard to their topographical relations, the contraction of the muscle to the electric stimulus is exceedingly irregular. Instead of the whole muscle responding, there are lacunæ which correspond to the bundles of fibres which have disappeared. After a time no electrical excitation, however strong, produces even a fibrillary contraction, for the whole parenchymatous tissue has been absorbed, and nothing remains but the interstitial connective tissue.

FIG. 60.



The reflex excitability in the early stages appears to be increased, but as the disease advances it becomes less and less, and is finally altogether lost. Thus when the fibrillary contractions, which characterize the initial period, are temporarily absent, they can be readily reëxcited, as previously mentioned, by striking the skin over the affected muscle.

Besides the paralysis, which it must be clearly understood results from the atrophy, and is directly proportional to its extent, there may be contractions. These, when present, are due to the fact that the atrophy has not attacked all the muscles of an extremity simultaneously, or to a like degree, and consequently, the normal antagonism lestroyed, distortions take place. When these occur in the hand, oduce the *main en griffe* of Duchenne. Of the twenty-nine ccurring in my experience, seven only had any distortions. In e paralysis, which is similar in several respects to progressive r atrophy, contractions and distortions are much more common.

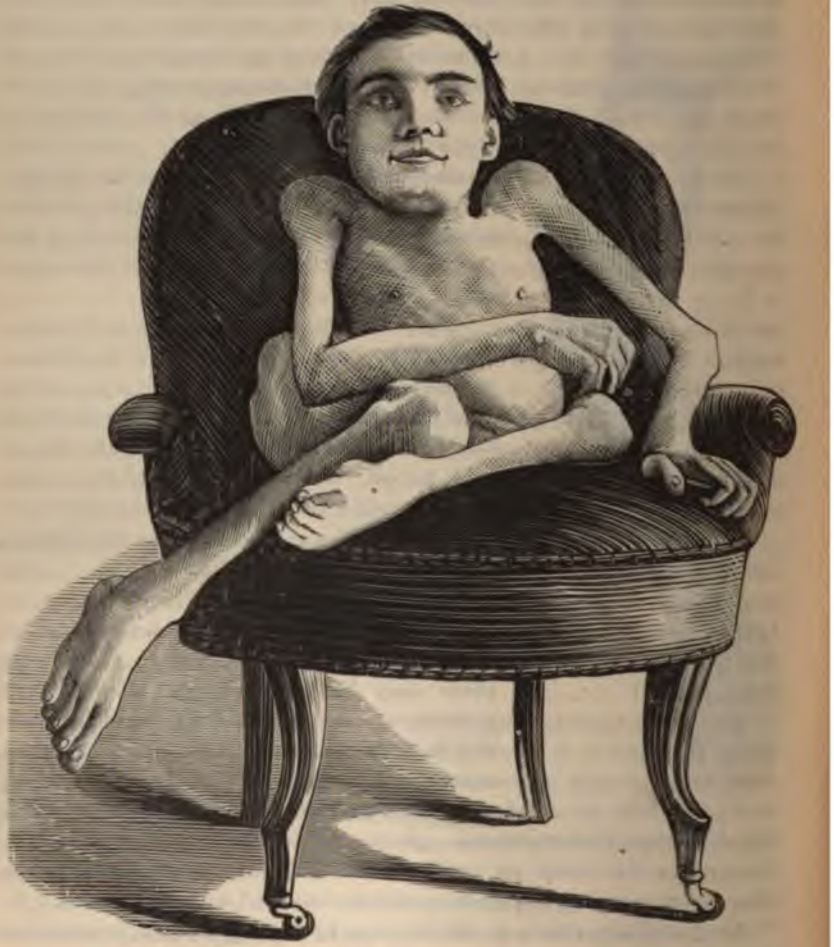
pupils are sometimes contracted from the implication of nerve- the cilio-spinal region of the cord. This was the case in one or es in four of my cases.

The course of the affection is slow, but in the great majority of cases it advances to a fatal termination. Death takes place from the muscles of respiration becoming involved, from exhaustion, or from some intercurrent affection. Several of my cases have lasted over ten years.

It is worthy of notice that up to the present time there is no instance on record of the muscles of the eye-ball or the levator palpebræ superioris being affected.

The accompanying woodcut (Fig. 61), from Friedreich, represents a patient, Ludwig Bessing, forty-five years old, who certainly presents a remarkable example of the disease. Almost all the muscles of the body, trunk and extremities, were in a state of extreme atrophy, the

FIG. 61.



only exceptions being found in the left forearm. The disease had remained stationary for many years, during all of which period there were strong fibrillary contractions. No hereditary influence could be ascertained to exist.

In a recently-published memoir, MM. Duchenne and Joffroy[1] have shown that glosso-labio-laryngeal paralysis is sometimes complicated with progressive muscular atrophy, and that this latter affection, implicating the muscles of the tongue, the lips, and the veil of the palate, has hitherto been confounded with the first-named disease. It differs from it, however, in the essential fact, which is applicable to the disorder appearing in other parts of the body, that the loss of power is not the initial symptom, but results directly from the diminution in the size of the muscles. This point will be further considered under the head of Diagnosis, when other cases similar to that here referred to will be brought forward.

The progressive muscular atrophy of infants presents some features different from those met with in adults. Duchenne,[2] who has elucidated this point of the subject, has ascertained that the initial atrophy, instead of beginning in the upper extremities, as it usually does, or in the trunk or lower extremities, as is occasionally the case, starts from certain muscles of the face, giving a very peculiar expression to the countenance. I have never witnessed, to recognize it, a case of progressive muscular atrophy in an individual under the age of eight years. Consequently, no instance of the infantile form of the disease has come under my notice. Duchenne has witnessed fifteen cases, and, in each, the beginning of the malady occurred between the ages of five and seven.

The muscle first to be affected is the orbicularis oris, and, as he states, its failure to contract occasions a characteristic thickness of the lips. The expansion of the mouth, as in laughing, is then only effected by the buccinator and the risorius. Eventually, other muscles of the face become involved, and finally the atrophy extends to the superior extremities, the trunk, and the lower limbs.

The accompanying cut (Fig. 62), after Duchenne, represents in profile the face of a boy thirteen years of age, whose lips had, in infancy, become thick and pendent, and whose orbicularis oris, levatores labii superioris, levatores labii superioris alæque nasi, and the zygomatici, had become atrophied, and, when stimulated by strong faradaic currents, gave no response. At the age of twelve the muscles of the chest had become affected.

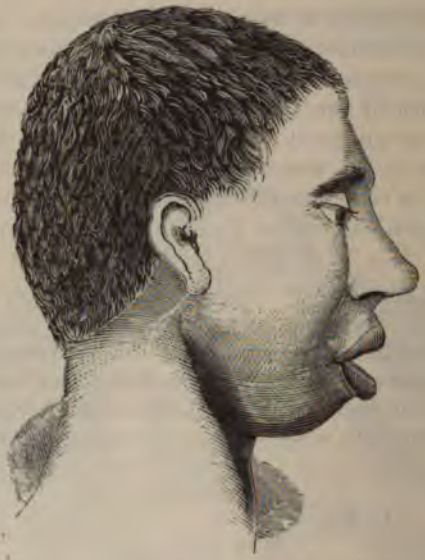
In this case, as in one other in Duchenne's experience, the disease had been transmitted through the mother, who was herself the subject of progressive muscular atrophy.

---

[1] "De l'atrophie aiguë et chronique des cellules nerveuses de la moëlle et de bulbe rachidien," *Archives de Physiologie*, No. 4, 1870, p. 499.

[2] *Op. cit.*, p. 518.

FIG. 62.



Causes.—Progressive muscular atrophy is not a disease of old age. Only two of my cases were in persons over fifty; four were between forty and fifty, and forty-six were under forty. Of these latter, three were between fifteen and twenty, and two between eight and ten. The period of life at which it appears to be most common is that extending from twenty-five to thirty-five.

Sex is a strong predisposing cause. All of my cases were in males, except one, a lady from Providence, Rhode Island, in whom the face and tongue were involved in the morbid process. Roberts[1] states that, of ninety-nine cases, eighty-four were males, and only fifteen females. Other authors have noted the greater proclivity of males. The difference appears to be due to the greater severity of muscular exertion required in many of the occupations of men.

Hereditary influence is a well-recognized predisposing cause. Two of my cases sent to me by Dr. Lincoln, of Washington City, were brothers, two others are sons of a prominent manufacturer of this city, and fourteen others had relatives affected with the disease.

But by far the most remarkable history of the hereditary transmission of the disease, which has come to my personal knowledge, is contained in the following account, which constitutes a pamphlet written by one of the unfortunate subjects, and sent to me by Dr. R. F. Andrews, of Gardner, Massachusetts. The interest attaching to the whole matter, as well as in consideration of the graphic, though homely, manner in which the story is told, will, I am sure, be sufficient apology for my quoting the entire pamphlet :

---

[1] "An Essay on Wasting Palsy," London, 1858, p. 135.

"MUSCULAR ATROPHY.

"Among my ancestors and their neighbors this disease was known as the 'Wetherbee Ail;' definitely, it is a wasting or consumption of the muscles. Until recently, it has been considered incurable; the cause is unknown, but generally the first intimation the patient has of it is a shock. My opinion is that its inception is some time previous, but not noticed. From and ever after the shock its progress and character are remarkable, the various symptoms and details of which will be seen in the individual cases I shall attempt to describe.

"I have been unable to trace the history of this disease beyond my great-grandfather, Ephraim Wetherbee, and all I know of his history is that he had six sons and two daughters, and that he died of the 'Wetherbee Ail.' His son Asa experienced a sensation in the calf of both legs, as if struck smartly with a whip; I do not know how long he lived, but he failed from that time; Isaac, another son, had the same disease, but I have been unable to learn any particulars in his case. Two others, Calvin and Joseph, the latter my grandfather, died in South America of diseases prevalent in that country; I can say nothing of the others. Hannah Wetherbee, one of the daughters, I can remember to have seen walk feebly and soon after confined to her room nearly helpless, and to have seen her coffin-lid screwed on. Sarah married a Mr. Paine; she had had seven children and was in good health; she was walking on the street and felt as if hit in the calf of the leg by a stone, and turned expecting to see the boy who threw it, but concluded that was not the case; she lost the spring of her toes, as she expressed it, and never walked naturally afterward; she told her family, on her arrival home, that she had the 'Wetherbee Ail.' She lived seven years, had the best of care and medical treatment; she had two children during her sickness, the last a son, after she had become perfectly helpless and only nine months previous to her death. She had nine children; one died young, the others are living and in good health. I had these particulars from the eldest, Sarah Paine, who married Spaulding, and is nearly sixty years of age, has generally been in good health, excepting during some three years she suffered from nervousness and lost all her strength; but she recovered and for some twenty years has been well. She had a son and daughter who both married; the daughter died of consumption of the blood, the son is in good health. Mrs. Spaulding names other cases but can give no particulars except that one felt the first shock in the foot under the shoe-buckle, such as were worn a hundred years ago; another was attacked in the brain and lived but twenty-four hours (I should not call that a case of muscular atrophy); another requested that an examination should be made after his death, which being done showed that all the muscles were consumed.

"Joseph Wetherbee, my grandfather, had a son and daughter; the daughter, Lucy, married a Mr. Pitts; she had only a son and daughter. The daughter lived some twenty years and died of some sudden and severe sickness. The son, J. Henry Pitts, is still living and is about forty-three years of age; has suffered much from rheumatic fever. Aunt Lucy herself, enjoyed good health till about fifty years old, when she died. She believed there was nothing peculiar in the so-called 'Wetherbee Ail.' Her last sickness was of an entirely different character.

"I now come to the case of my father. He was of a robust build, had a strong constitution and was temperate, drinking no spirits since my remembrance, probably not much before; used some tobacco at times, and worked hard at different trades, as shoemaking, farming, and chair-making. When about thirty-nine he remarked that he was growing old fast, and some of the neighbors discovered a slight limp in his walk. I was not living at home at that time, and do not know much of his condition in the early stages of his sickness. He first discovered a weakness in the right thumb, being unable to open his pocket-knife in the usual way. The right hand and arm lost strength faster than the left; and, contrarily, the left leg failed the fastest. He thought the direct cause of his lameness to be over-exertion in harvesting a crop of meadow-hay, in August or September, 1844. He continued to labor about a year. The progress of the disease was rapid; he suffered somewhat from painful muscular contractions or cramps, otherwise he had but little pain. The larger muscles of the arms and legs became soft and flabby, and diminished in size. In November, 1845, he cut his fingers in the shop, went home and never entered the shop again. He got about the house with crutches several months, comfortably. During the following winter he had rheumatic fever. In the summer of 1846 he became nearly helpless. Mother and myself lifted him to his feet, and to and from his bed and chair. The kidneys were also affected, and the lungs were very weak. So he wasted in flesh and strength, and died on the 10th of October, 1846, a little more than two years after the hard work in the meadow.

"I can say no more of the above cases, except that the persons were native-born Americans. Mrs. Spaulding thinks we descended from the English. I do not learn that there was dissipation in any branch of the family. There are branches of the family in which nothing of the kind appears; there is nothing of it in the Wetherbees in Scotland. Mrs. Spaulding thinks the disease was in the family previous to the time of my great-grandfather.

"I was born on July 23, 1831, in Westminster, Massachusetts. At the age of five I was thrown from a wagon and got a scalp-mark from the horseshoe. At the age of six I remember an aching head and discharge at the ear; at seven or eight a bad cold, with soreness of chest, a cough and hot gin-sling, none of which were in the least agreeable. When eleven I was badly poisoned with ivy, although before that I had handled it with impunity; at fourteen another cold and affected chest and lungs, with ulcers, or something like, in the head.

"From this time to the age of twenty-one I had some sick-headache; got sick three times from trying to paint outside work, got poisoned with ivy and dog-wood, but did not lose many meals or much sleep. I worked at chair-making and had no lack of out-door exercise. As I have spoken of shocks being felt by some of the above-named persons, I am reminded that I felt one on a day in the summer that I was sixteen; I felt as if struck with a piece of board on the left shoulder, head, and neck. I looked around for the cause but saw no one; I was not hit nor hurt; have felt something similar since, but as nothing came of I thought no more about it. At twenty-one I had a lame stomach, partly from work and from getting a blow in the breast. One plaster set that all right, and I have had nothing of it since.

"In January, 1855, I had lameness in the right wrist and hand, attributing it to a slight, and at the time unnoticed, sprain by rolling logs. I had much pain and trouble during three or four years; many times I could scarcely write, and came to use the hammer and saw with the left hand. It is useless to name the various modes of treatment, as time only seemed effectual in restoring the parts to nearly their natural condition. In December, 1855, I had a severe cold, affected lungs and head; had discharges at the ear, but kept the house for a few days and recovered. Early in the summer of 1857 I had poor appetite and no ambition, headache, and slight night-sweats. I gave up work early in August, put myself under a doctor's care, improving much in two months, and before winter gained my usual health. Early in the spring of 1858 I had palpitation of the heart, caused by eating new maple-sugar; have been subject to it ever since, at intervals from a week to a year and a half, always brought on by drinking water or ale, or eating an apple. I felt somewhat weak during their continuance, but usually kept at what I happened to be doing, though they lasted from six to thirty-six hours. Two or three years following I had two sharp stitches in the back, by lifting a slight weight, resulting in a lame back for a season.

"In August, 1862, I enlisted in the army and soon went to Virginia; had but little difficulty in getting accustomed to camp-life and climate. I had no occasion to answer surgeon's call until the following winter, when I got cold, being on guard night and day during Burnside's so-called 'mud march,' resulting in pain in the bowels and diarrhœa, but that all wore away in a month or two. During May, 1863, while in camp at Washington, I took a cold which troubled me till after the battle of Gettysburg, when I had my left thigh fractured by a spent grape-shot. The fracture, only a simple diagonal, was never set, the bone uniting in its own way and time, consequently the left leg is about two inches shorter than the other, and crooked.

"In 1864 I received my discharge. I walked with a cane the following summer, then gave it up; experienced no difficulty excepting the limp resulting from the shortness of the leg. During the year 1865 I was engaged in work which kept me on my feet. I frequently walked two miles, out and back, but experienced more fatigue than in previous years. In May, 1866, I went to Chicago, and engaged in sedentary occupation; had about seven-eighths of a mile to walk to and from work. I usually walked rapidly, many times beating the horse-cars. In July, 1867, I went to Pennsylvania and engaged in chair-making; on my feet all of the time, and some of the time standing still at a machine; also walked much over the rough hilly roads in that country. I was there upward of three years; during the time I had two or three attacks of lame back, also of piles. When taking an armful of stock from the floor I found it convenient to keep a stick in the right hand to assist in rising. In a letter to my brother I remarked I felt that I was getting old. As I was then about the same age my father was when he made the same remark, the coincidence is remarkable.

"Late in 1869 or early in 1870, I noticed a fibrillous contraction just above the right knee, about half-way from the anterior to the inside. It is a tremulous twitching of the muscles, which is seen in the muscles of slaughtered animals after the skin is taken off. It is painless but somewhat disagreeable, and more noticeable after retiring. In two or three days it was gone. I was at that time standing at a lathe during the day, and walking rapidly morning, noon, and night. In November, 1870, I came to Gardner, where my employment was such that I had to stand stiller than ever. I was advised to sit on a stool part of the time, but I was not inclined to do so. I walked rapidly to and from the shop, each trip requiring about twelve minutes, four trips a day up hill and down. In February, 1871, I felt a general lameness or muscular soreness from over-exertion, loss of sleep, and taking cold. I had had such experiences before, most persons have the same. About the 20th or 25th of March I noticed for the first time that I went up-stairs with much difficulty, the trouble seeming to be in the right thigh. On the 26th of March I walked to Westminster, a distance of five miles, and back. I felt generally fatigued, but noticed no particular lameness. That was the last foot-journey of any distance I ever took. Twice in April I quickened my pace to pass some persons on the sidewalk, and felt a quick, painful sensation in the anterior portion of the right thigh. A few days subsequent to this there was an alarm of fire about twelve o'clock. I started to run, but gave it up after a few steps, and have not tried to run since.

"I was a little anxious about all this, but did not suspect any permanent lameness existed. I cannot say when the thought of the 'Wetherbee Ail' came into my mind. On May 11th, as I was going of an errand in the morning, I stopped to throw a stone at a small hawk in an apple-tree, but fell myself, and the hawk flew away. Twice soon after I fell on throwing a stone. About this time I had severe cramps in the right thigh, and have the impression that there were cramps in the right thumb. I consulted a doctor about this time, and begun a regular course of treatment. The 1st of June I gave up going home for my dinner, and sat on a stool much of the time while at work. I still walked comfortably, but could not raise my weight up an ordinary step, and I had to be careful that the knee set at every step, or it would cripple and let me down. The reader will notice that the leg which was not broken failed; as it was some two inches longer than the other, it had to bear the greater burden, and, in going up-hill or up-stairs, virtually had to raise my weight two inches higher at every step than the broken leg. The tremulous twitching was very marked during this time, and occasional painful cramps. I continued to lose strength all summer, and was obliged to give up some kinds of work.

"September 1st, I found that the right thigh measured only sixteen inches, while the left measured nineteen. I used a cane at this time and found it of much service. At this time I rode to and from the shop. About the 1st of November I found the left leg began to fail. The 1st of January, 1872, I found much difficulty in walking only a short distance. I gave up work and went to the Massachusetts General Hospital for five weeks; but no effect of the treatment was apparent; think the right thigh had decreased to fourteen and one-half inches in one year. There was but very little strength in the right leg, the muscle of the thigh was very flabby, and the heat was lower than in the other leg. I resumed my occupation in February. The fibrillous contractions and painful cramps had by this time nearly ceased in the right leg, but were visible in the left; also noticed weakness in the right thumb, especially when cold, and could not hold a carpenter's pencil in the usual way. I cannot state the number of times that I fell; I continued to ride to my work. Late in May I could bear no weight on my left toes. Meantime I had bandaged my right foot and leg to the knee, on account of swelling. On the 27th of July I was thrown from a carriage, the immediate result of which was a general muscular soreness, particularly in the left foot and arms.

"From this time the progress of the disease was marked and rapid, especially in the ball of the right thumb and the left thigh. I now found it unsafe to step without a cane, or out of the reach of something permanent. As the cold weather came on, I had to change my job for a lighter one in a warmer place. I gave up walking in the shop, used a wheel-chair, and was during the fall put into a buggy by a strong man. I continued to work through December, excepting some of the coldest days, but on the last afternoon of the year 1872 I fell and severely sprained the left knee; was obliged to quit work, and have done nothing since.

"At no time, since I first felt the lameness in the right thigh, have I been able to say with truth that I was a little better, or even about the same; but that I was not so strong as I was a month previous. This disease never stands still. I will close this sketch by saying, that at this writing I cannot stand alone, have no control of the right leg whatever, and cannot move the toes. The left is very weak; both feet, and the legs below the knee, are somewhat cold most of the time. I dress without assistance. My arms are not strong enough to raise my weight to my feet; have not strength to cough or sneeze with any force. Have a fair appetite and sleep well. It is probable that nearly every muscle in the system is affected, as I have felt the cramps and tremulous contractions in nearly every part. There is no loss of sensation in any part. The large muscles of the right thumb are much wasted, the whole hand has a bony appearance, and the third finger droops. Sometimes I cannot pick up a pin, and my writing is scarcely legible. I gave up all treatment six months ago, as I could never see any difference in the progress of the disease while under treatment and while not.

"One theory is, that this disease is not inherited by the descendants of the females; and the history of Mrs. Paine's family seems to confirm it. My object in writing this is, that those into whose hands it may fall, who are predisposed to this disease, may keep a watch upon themselves; and I exhort them to moderation in labor and physical exertion, and in all things, and that they may have a history, though imperfect, of the cases which have appeared in our family; that they may immediately, on suspicion that they are attacked by any unusual thing, apply for the best help within their power. I waited some two months before taking any measures for relief. This disease is also known as 'wasting palsy.' It is known in other families. The so-called living skeletons, who are exhibited as curiosities, are sufferers from muscular atrophy in its worst form. I am the oldest of five; one sister and three brothers still enjoy fair health. None of us have used tobacco or spirituous liquors.

"E. H. WETHERBEE.

GARDNER, MASSACHUSETTS, *March* 31, 1873."

In relation to this case, Dr. Andrews, in sending me the pamphlet, writes, under date of March 30, 1874:

"This man, Wetherbee, died December 23, 1873.

"His sister has recently consulted me with symptoms of the same disease. The left arm and shoulder are affected. The twitching of the fibrillæ is worse at night. I prescribed iron and quinine, and rest. I was present at your clinic at Bellevue two years ago when you exhibited a patient—a bridge-builder—from Ohio, with the disease.'

Two members of this family, within the knowledge of the writer of the pamphlet, were affected with progressive muscular atrophy, and it is probable that other members, as he was informed, before his great-grandfather, were its subjects. An interesting circumstance is, that two of the cases were females, and it is likewise a notable fact that the children of one of these, nine in number, exhibited no symptoms of the disease. We have seen that in pseudo-hypertrophic spinal paralysis the affection is only transmitted through the females, while progressive muscular atrophy, so far as this history goes, appears to be only immediately hereditary through the males. Atavism was therefore manifested in a different way than it is in the former affection. Duchenne, however, as we have seen, has witnessed two cases occurring in children in which the disease was transmitted directly through the mothers who were themselves the subjects of the malady.

But nothing on this point can surpass the instructive histories related by Dr. Naunyn[1] relative to cases brought before his medical clinic in Königsberg. Six generations were subject to the disease. Members of three generations were alive at the time Naunyn delivered his lecture, and the clinical histories of seven cases were personally known to him. The oldest of these, Dorothea Braun (*née* Bessel), was seventy years old. Her father and grandfather had the affection, to her knowledge, and her father told her that her great-grandfather was also its subject. Dorothea had eleven brothers and sisters, of whom only one, Minna, a sister, had the disease. Of her own seven children four were affected. Of Minna's three, one dying in early infancy, two were diseased. Of Dorothea's uncles, seven in number, two suffered from the malady.

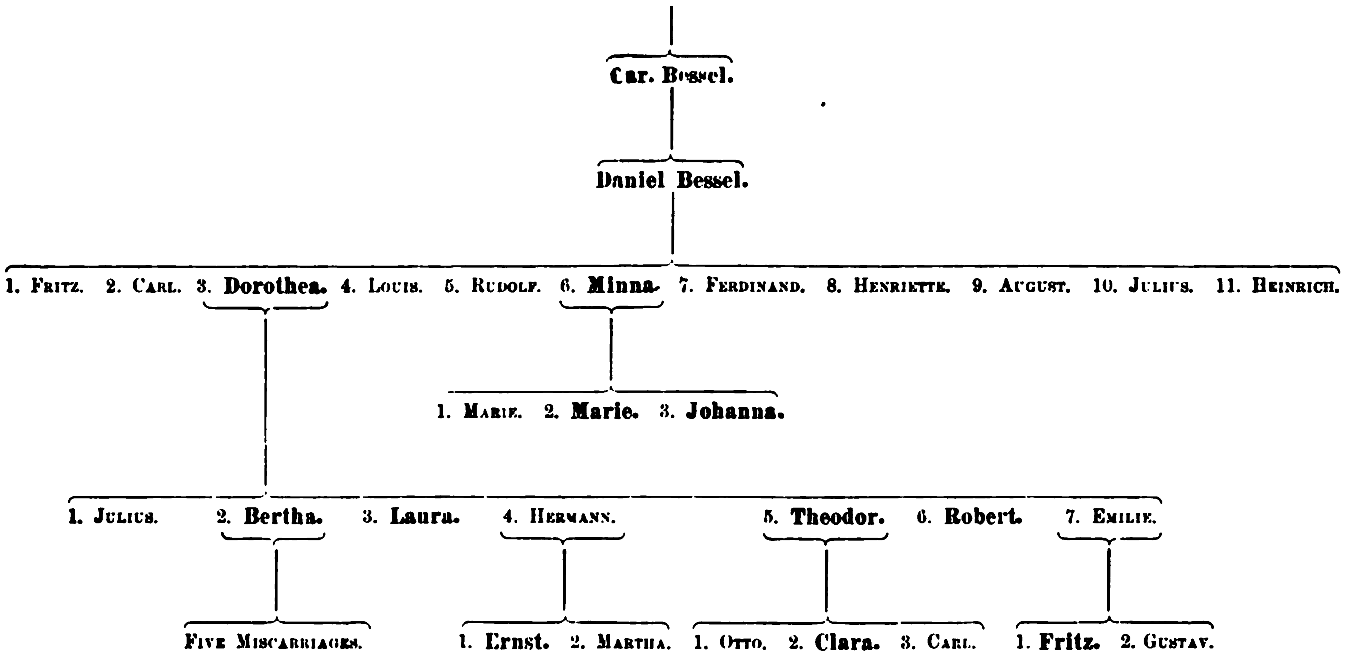
The accompanying table shows at a glance the relationship of the several members of this remarkable family, and the channels through which the disease was transmitted directly, and by atavism. From its examination we see—

1. None of the sons of Daniel Bessel were affected, but two of the daughters, Dorothea and Minna, were, and the malady was propagated by them.
2. Of Minna's three children, all females, two had the disease.
3. Of Dorothea's seven children, two boys and two girls were affected, while two boys and one girl escaped.
4. One of the boys, Hermann, and one of the girls, Emilie, who escaped, had each a boy who had the disease, thus affording two examples of atavism, one through the male and one through the female.

This history is in marked contrast to that of Wetherbee so far as the line of descent is concerned, and the two together may be considered as definitely settling affirmatively the question of the hereditary transmission of progressive muscular atrophy.

---

[1] "Ueber Heredität der progressiven Muskelatrophie," reported by Dr. Eichorst in *Berliner klinische Wochenschrift*, Nos. 42 and 43, 1873.


Car. Bessel.
Daniel Bessel.
1. Fritz. 2. Carl. 3. **Dorothea.** 4. Louis. 5. Rudolf. 6. **Minna.** 7. Ferdinand. 8. Henriette. 9. August. 10. Julius. 11. Heinrich.
1. Marie. 2. **Marie.** 3. **Johanna.**
1. Julius. 2. **Bertha.** 3. **Laura.** 4. Hermann. 5. **Theodor.** 6. **Robert.** 7. Emilie.
Five Miscarriages.
1. **Ernst.** 2. Martha.
1. Otto. 2. **Clara.** 3. Carl.
1. **Fritz.** 2. Gustav.

The individuals whose names are printed in heavy-faced type were the subjects of the disease—the others escaped.

From some facts which will be adduced under the head of treatment there is reason to believe that syphilis is occasionally a cause of progressive muscular atrophy.

The exciting cause is often impossible of detection. This was the case in twenty-nine of the instances that have come under my observation. Of the remaining twenty-three, injuries of the spine were the cause in two, exposure to cold and dampness in thirteen, and excessive muscular exertion in eight. Of these latter cases, two occurred in the persons of ballet-dancers, the disease making its appearance first in both gastrocnemii muscles simultaneously; one in a gentleman who had overtasked the muscles of the upper extremities by severe and long-continued exertion in rowing—the muscles about the shoulders being affected; in two, the muscles of the right hand were first attacked, as the result of excessive use of the pen in writing; in one, it was induced by the occupation, that of a bricklayer, requiring the patient to bear the weight of his body, during his work, mainly on one leg—the one attacked; in one, it was apparently induced by running a long distance; in one, it began in the thenar eminence of the right hand of a bridge-builder; in one, it attacked the muscles of the hand and forearm, beginning in the ball of the thumb in a man whose occupation—faro-dealer—required him to use his thumb and fore-finger in a peculiar way for many hours at a time. Venereal excesses have been alleged as a cause, but I have seen nothing to support the assertion.

Diagnosis.—Progressive muscular atrophy may be confounded with infantile spinal paralysis, spinal paralysis of adults, pseudo-hypertrophic spinal paralysis, amyotrophic lateral spinal sclerosis, and various secondary forms of atrophy.

From all these diseases it is discriminated without difficulty, if attention be paid to its peculiar features, which in the main are as follows:

1. The absence of fever and of pain in the back.
2. The gradual progress of the atrophy, the muscles being attacked one by one and not *en masse*, as in the other diseases named.
3. The fact that there is not paralysis in the proper sense of the word, the loss of power being simply the result of a diminished mass of muscle.
4. The retention of the electric contractility so long as there are muscular fibres to contract.
5. The presence of fibrillary contractions, which are very rarely met with in other atrophic diseases, except amyotrophic lateral sclerosis, the diagnosis from which will be herewith pointed out.

Progressive muscular atrophy, when manifested in the tongue, has often been mistaken for glosso-labio laryngeal paralysis. It is readily distinguished, however, from this latter disease by the fact that atrophy is not an accompaniment of the morbid process which characterizes glosso-labio laryngeal paralysis. In progressive muscular atrophy attacking the tongue the organ is marked by knots and depressions, the latter corresponding to the situation of the atrophied muscular bundles and the former to the as yet untouched portions. In glosso-labio laryngeal paralysis the tongue lies motionless in the mouth, undiminished in size.

In locomotor ataxia there is sometimes a wasting of the muscles, but the fact that the atrophy is shown in masses of muscles at once, and the clinical history of the patient, will suffice to render the diagnosis exact.

In rheumatic affections there is often atrophy, but this is consecutive on paralysis, and in the cases of tumors of the cord we have the phenomena of slow compression in addition to those of muscular atrophy.

In cases of injury of the cord or of the nerves supplying a part, paralysis is the first symptom to make its appearance, though atrophy may very quickly follow. In such instances the electric contractility is soon lost. Attention to the clinical history of such cases will render a mistake in their diagnosis almost out of the question.

Prognosis.—From what has been said, it will readily be apprehended that progressive muscular atrophy is a very serious disease; indeed, it is one of the most progressive of all the affections to which the term has been applied.

In only three cases have I succeeded in arresting the course of the disease, and in restoring the atrophied muscles. One of these was that of a highly-intelligent gentleman, formerly an officer in the navy, but now a resident of this city, whose case has already been referred to as having been induced by rowing; the other was that of the patient, also previously mentioned, in whom the affection was induced by cold, and which began in the right deltoid muscle. Both of these patients were entirely cured, regaining full muscular power. The other was a man who came to my clinic at the University Medical College during the winter of 1874–'75.

In four other cases, which I saw before the disease had advanced to a great extent, its progress was arrested, but there has as yet been no restoration of the wasted muscles; in two of these there was no probable cause of the affection.

The coexistence of a clinical history of syphilis probably makes the prognosis more favorable than would otherwise be the case.

The existence of an hereditary tendency renders the prognosis much more grave; and the fact of the disease having lasted a long time is also of unfavorable import.

Morbid Anatomy and Pathology.—Investigations in regard to the morbid anatomy of progressive muscular atrophy relate to the condition of the spinal cord, the nerves, and the affected muscles.

The spinal cord has been examined in cases of progressive muscular atrophy by Bergmann, Meryon, Gull, Luys, Lockhart Clarke, and others, with very different results; some of these observers finding no change whatever, and others detecting notable variations from the normal structure. In three cases examined by Clarke,[1] disorganization of the spinal cord, especially of the gray matter, was found, with, in one case, deposit of amyloid corpuscles.

More recently Hayem,[2] and Charcot and Joffroy,[3] have studied the morbid anatomy of progressive muscular atrophy with great care. In Hayem's case, the disease affected the muscles of the upper extremities to such an extent as to render them powerless from the shoulders down. The patient died from paralysis of the diaphragm, and of pneumonia.

On post-mortem examination, the spinal cord appeared healthy to the naked eye. The anterior roots of the cervical nerves were, however, notably atrophied. The most attenuated were those of the second, third, fourth, and fifth pairs. The sympathetic was healthy. On microscopic examination of the cord, the most marked characteristic was atrophy and disappearance of the nerve-cells. In some portions there were none to be seen, but there were large numbers of free nuclei, and of cells containing many nuclei. The atrophy of the nerve-cells, and of the anterior cornua of gray substance, was greatest at the level of the second and third cervical nerves, and extended as low as the fifth cervical. This region was that from which the nerves supplying the atrophied muscles were derived. In the dorsal and lumbar regions there was no atrophy of nerve-cells or of nerve-roots.

A consideration of this case shows, as Hayem remarks, that it is one which, during life, exhibited the usual symptoms of progressive muscular atrophy, and that, at the post-mortem examination, lesions were found in the muscles in the anterior roots of the nerve, and, above all, in the spinal cord. The alterations from the healthy structure of the cord consisted of—

1. Abnormal vascularization with dilatation, and sclerosis of the arterioles, and of the larger capillaries.

2. A more or less abundant exudation surrounding the blood-vessels.

3. Multiplication of the elements of the interstitial tissue (the neuroglia), and finally atrophy, and disappearance of a very great number of the nerve-cells.

---

[1] Beale's "Archives of Medicine," vol. iii., 1861; also, same, vol. iv.; also, *British and Foreign Medico-Chirurgica. Review*, vol. xxx., 1862.

[2] "Note sur un cas d'atrophie musculaire progressive, avec lésions de la moëlle," *Archives de Physiologie*, No. 2, 1869, p. 221, and No. 3, 1861, p. 391.

[3] "Deux cas d'atrophie musculaire progressive, avec lésions de la substance grise et du faisceaux antéro-latéraux de la moëlle épinière," *Archives de Physiologie*, Nos. 3 and 5, 1869.

These facts point to the existence of chronic inflammation of the gray substance of the cord, beginning in the nerve or parenchymatous tissue, and subsequently involving the neuroglia or interstitial substance.

The two cases of MM. Charcot and Joffroy have also been very carefully and thoroughly studied.

The chief features of the first case were, progressive muscular atrophy, especially marked in the superior extremities; atrophy of the muscles of the tongue and of the orbicularis oris, and paralysis with rigidity of the inferior extremities. The patient was a woman, and, becoming suddenly very weak, died asphyxiated.

At the autopsy, the anterior roots, especially those of the cervical region, were found greatly atrophied and discolored. The cord appeared healthy to the naked eye, except that at the dorso-lumbar enlargement it was softened. On microscopical examination, however, the nerve-tubes of the anterior columns were discovered to be atrophied, a great number being only represented by the axis cylinder, while the connective tissue was very much increased. The posterior columns were not involved in the least.

In examining the gray substance of the cervical region, the authors were struck with the extreme degree of atrophy which the cells of the anterior cornua had undergone; a large proportion of them had entirely disappeared, leaving no trace behind them. The posterior cornua appeared to exhibit all the qualities of the normal condition.

The alterations in the other regions of the cord were not directly connected with the muscular atrophy, except as regards the medulla oblongata, where the cells of the nuclei of origin of the hypoglossal were found to be atrophied, and even completely destroyed. In the second case, similar structural changes were found.[1]

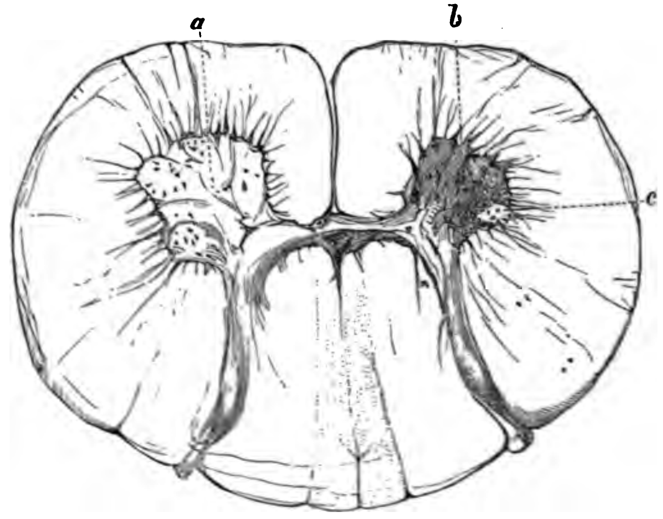
As Charcot states, when the alterations of the neuroglia are very pronounced, the anterior horn, which is the seat of the morbid process, may be considerably reduced in size. This condition is well shown in the accompanying woodcut (Fig. 63), which represents a section of the spinal cord taken from the cervical region of a patient who had been the subject of progressive muscular atrophy—*a*, the left anterior horn of gray matter; *b*, the right anterior horn, the cells of which are atrophied with the exception of a small group at *c*. The whole right anterior horn is seen to be diminished in size.

---

[1] These cases, which at the time were considered to be instances of progressive muscular atrophy with complications, are now to be classed under the head of amyotrophic lateral spinal sclerosis. I have described here the morbid anatomy exhibited by them in so far as it relates to the lesion of the cells in the anterior horns of gray matter, reserving the consideration of the other lesions for a subsequent division of the subject. It may be said now, in anticipation of a fuller discussion, that the alterations of the gray matter of the anterior horns appear to be the same in the two diseases.

MM. Prévost and David[1] have recently reported a case of atrophy of the thenar eminence, similar to that related on page 521, as occurring in my own experience. They had the opportunity, however, of making a post-mortem examination, the patient dying of a wound of the head. The man, the subject of the disease, had had from his infancy complete atrophy of the muscles of the ball of the right thumb. Even the bone was atrophied. There had never been pain.

FIG. 63.



On post-mortem examination there were found: manifest atrophy of the anterior root of the right eighth cervical nerve; slight atrophy of the anterior root of the right seventh cervical nerve, and atrophy of the right anterior horn of gray matter in relation with these roots. The muscles of the thenar eminence were entirely destroyed; but all the other muscles of the hand and arm were normal.

In this case the relation between the spinal lesions and the affected muscles was sufficiently explicit.

Still more lately MM. Pierret and Troisier[2] have examined the spinal cords of two patients who died of progressive muscular atrophy, and have confirmed in all essential respects the results obtained by the observers previously mentioned. The character of the lesions of the cord and nerves may therefore be considered as definitely ascertained; and it is equally an established fact, first noticed by Cruveilhier, that the anterior roots of the spinal nerves derived from the affected portion of the cord and supplying the diseased muscles are generally found atrophied from the disappearance of a certain number of nerve-tubes. This is a secondary lesion resulting from the spinal degeneration.

---

[1] "Note sur un cas d'atrophie des muscles de l'éminence thénar droite avec lésions de la moelle epinière." *Archives de Physiologie*, 1874, p. 593.

[2] "Note sur deux cas d'atrophie musculaire progressive," *Archives de Physiologie* 1875, p. 257.

The atrophy of the muscles is due to the degeneration and ultimate disappearance of the fibrillæ. To the naked eye they appear pale and attenuated. By microscopical examination, it is seen that the transverse striæ of the fibrillæ are in course of disappearance, and as the disease advances they are perceived to fade away altogether. Eventually, the longitudinal striæ also disappear. At the same time, the muscular fibrillæ break up into granules, and then undergo regressive metamorphosis into fat. It is not uncommon to see a bundle of fibrillæ, in one part of which the transverse striæ only have vanished; in another, the longitudinal; in another, the process of disintegration complete; and in another, oil-globules occupying their place. Fat-corpuscles are frequently found deposited between the bundles of fibrillæ. After a time the fat disappears, and nothing is left of the muscle but a cord of connective tissue made up of the perimysium.

Sometimes the interstitial fat is deposited in such large amount as to take away from the atrophied parts all appearance of emaciation, and, in fact, to mask the essential feature of the disease. Duchenne has particularly called attention to this circumstance, and has given engravings representing patients thus affected. This fact, it appears to me, furnishes an additional argument to those previously advanced relative to the propriety of placing pseudo-hypertrophic spinal paralysis in the present group of diseases; and Charcot, who considers progressive muscular atrophy an affection of the cells in the anterior horns of gray matter, but regards pseudo-hypertrophic spinal paralysis as a malady of the muscles, speaks of the excessive lipomatosis—*lipomatose luxuriante*—of the former disease, without appearing to recognize the fact as indicating any relationship between the two.

The essential points in the morbid anatomy of progressive muscular atrophy are no longer matters of doubt. The bearing of these points on the real nature of the disease is next to be investigated.

At the outset of the inquiry relative to the pathology of progressive muscular atrophy, the question arises, Is it an affection of the muscles, the nerves, the sympathetic system, or the spinal cord?

As regards its being a disease primarily of the affected muscles, Friedreich[1] is the most strenuous contestant in support of the affirmation. His main argument is that lesions are found in the muscles while they are not found in the spinal cord or nervous system, except in a few instances. But he neglects to state these very important facts, that in every case he cites, in which lesions of the cord were not found, the examination was made before Lockhart Clarke had taught us how histological investigations of the nervous centres were to be carried on, and that in every case of progressive muscular atrophy, in which the spinal cord has been examined since that time, and according to that method, disease of the anterior tract of gray matter has been found. Thus the first examination which he cites was made in 1858; the last in 1867. In the intervening period the lesions of the cells in the anterior horns did not attract attention—were not, in fact, discovered. Lockhart Clarke, Charcot, Joffroy, Duchenne, Hayem, Pierret, Prévost, and others, had not made the examinations which have placed the existence of the central lesion beyond a doubt.

---

[1] "Ueber Muskelatrophie," u. s. w., Berlin, 1873.

Now, as to the relation of cause and effect which the spinal and muscular lesions bear to one another, opinions vary, and the question appears to be one which, in its very nature, is incapable of being positively solved. We can only take the evidence on both sides, and determine the matter according to what strikes us as being the weight of testimony; and this appears to be in favor of the doctrine of primary spinal disease. We have in support of this view—

1. The fact that those cells of the cord are diseased which are in anatomical and physiological relation with the affected muscles.

2. The absolute certainty that similar lesions of the anterior horns of gray matter will cause atrophy of muscles —infantile spinal paralysis, spinal paralysis of adults, pseudo-hypertrophic paralysis, acute myelitis, etc. In these diseases we know from the central as well as from the peripheral phenomena that the morbid process starts from the spinal cord. We have hence evidence that atrophy of nerve-cells will give rise to atrophy of muscles.

3. On the contrary, we have nothing to show that atrophy of a muscle will cause inflammation and degeneration of spinal nerve-cells.

4. If the disease were a primary affection of the muscular system, we ought to find the nerves diseased at their extreme peripheral terminations in the muscles; such, however, is not the case. The ascending neuritis, which Friedreich assumes to exist, is not shown to be a pathological entity. Neither the patho-anatomical facts nor the symptoms of progressive muscular atrophy give any color of truth to his theory.

It is not to be doubted, however, that peripheral lesions of the nervous system will cause central disease. But we can readily concede that much, without going to extreme lengths with Friedreich.

But no one can properly study the question without perceiving the manifest inconsistency of Charcot and others in contending for the central origin of progressive muscular atrophy while denying such a beginning to pseudo-hypertrophic spinal paralysis, an affection which presents so many analogous features to the first-named disease both in its symptomatology and its peripheral and central lesions. Here Friedreich is supported by the eminent French pathologist.

As to the affection being a primary disease of the nerves, the only evidence we have of that doctrine is the fact of the atrophy of the anterior roots of the spinal nerves in direct relation with the atrophied muscles. Cruveilhier regarded this condition as the essential lesion, mainly, however, because he was unable with his imperfect means of research to discover the morbid process in the cord. This nerve-atrophy is like that of the muscles—to be regarded as entirely secondary to the central disease, and as being directly dependent thereon. If it were primary or due to the muscular atrophy, we would find it not only manifested in the anterior nerve-roots but in the peripheral extremities; beginning in them and passing along the trunks of the nerves to the cord.

When we come to consider the relation of progressive muscular atrophy to the sympathetic nervous system we find little or nothing to warrant us in considering it as one of cause and effect. It is true that Jaccoud[1] and others have observed lesions of the sympathetic, associated with the disease in question; but Charcot, Vulpian, and Hayem, by the employment of the most approved methods of research, have failed to confirm these results; and quite recently M. Lebimoff[2] has most thoroughly and conclusively, in a case of undoubted progressive muscular atrophy, investigated the sympathetic nervous system, and has found neither fatty degeneration of the nervous element nor degeneration or proliferation of the neuroglia. All that he discovered was a deposit of pigmented granulations in the protoplasm of the connective-tissue cells—a condition which he very properly ascribes to the general exhaustion and the cachectic state of the patient. In this case the characteristic alterations of the cells of the anterior horns were very pronounced.

Hence we are, I think, forced to conclude that progressive muscular atrophy is not primarily a disease of the muscles, the nerves, or the sympathetic system, but of the anterior tract of gray matter of the spinal cord.

As to the nature of the process by which the cells are destroyed there is every reason to believe that it is a very slow, chronic inflammation.

Relative to the physiological functions of the cells which are the seat of the disease, there is not much to say in addition to the remarks already made when infantile spinal paralysis and spinal paralysis of adults were under consideration.

Progressive muscular atrophy, pure and uncomplicated, is unaccompanied by paralysis, except such loss of power as is directly due to the diminution of the volume of the affected muscles. The inference is, therefore, that it is not the motor cells which have disappeared or become atrophied, and yet, on post-mortem examination, we find that nerve-cells of *some* kind have been diseased. The presumption is, and it is reasonable, that these are cells which are specially connected with the nutrition of muscles—trophic cells—and that progressive muscular atrophy is a symptom indicating the existence of disease of the trophic cells. The very existence of these cells is a matter of inference, but in my opinion the argument in favor of the affirmative is very much strengthened by the facts furnished by the morbid anatomy of progressive muscular atrophy. Dr. Handfield Jones[1] has recently written forcibly against the existence of any special trophic nerves, and, by extension of reasoning, trophic nerve-cells. But he was unaware of the more recent researches of Duchenne and Joffroy,[2] upon which, in accordance with these observers, I have based my views of the pathology of progressive muscular atrophy, and to which I have already alluded. We have only to take into consideration the phenomena which are exhibited in glosso-labio-laryngeal paralysis as it affects the tongue and progressive muscular atrophy attacking the same organ, to perceive how wide is the difference between the two affections. In the case of a lady from Rhode Island, now under my care, the thenar eminences of both hands, certain muscles of the arms, and others of the lower extremities, are in a state of profound atrophy. One side of the face is also affected. She swallows with difficulty and speaks with great indistinctness. Here are some of the symptoms of glosso-labio-laryngeal paralysis to a superficial observer, but when the patient opens her mouth the tongue is seen not as a mass of reddened, flabby, inert muscles lying torpid, but atrophied to a marked degree on the left side and capable of being moved as well as the diminished volume of muscular tissue will permit. Here we have atrophy of the muscular system beginning in the upper extremities and finally attacking—still preserving its characteristics—the muscles of the face and tongue.

---

[1] "Bulletin de la société médicale des hôpitaux," 1864; and, "Traité de pathologie interne," tome i., 1870, p. 357.

[2] "Recherches sur l'état du système nerveux sympathique dans un cas d'atrophie musculaire progressive spinale protopathique," etc., *Archives de Physiologie*, 1874, p. 889.

On the other hand, we may have the morbid process, which gives rise to glosso-labio-laryngeal paralysis, extend down the cord and attack the cells of the anterior horns. But it is then a paralysis which results, not an atrophy, and the lesions of the anterior horns are to be classed with the secondary degenerations of the cord.

Are we not, from these two categories of cases, still further warranted in assuming the existence of motor and trophic cells both in the spinal cord proper and the medulla oblongata? To answer this question in the negative it appears to me we are forced to disregard some of the most cogent teachings of morbid anatomy and pathology.

---

[1] "Are there Special Trophic Nerves?" "St. George's Hospital Reports," vol. iii., 1868, p. 89.

[2] "De l'atrophie aiguë et chronique des cellules nerveuses," etc., *Archives de Physiologie*, No. 4, 1870, p. 499.

Treatment.—The most approved means of treatment consist in the use of the primary or galvanic current to the spine, and the faradaic to the atrophied muscles. The former is best applied by placing one pole on the nape of the neck and stroking the skin on each side of the vertebral column with the other. The current should be as strong as the patient can endure. A *séance* should be given every alternate day, and should last about ten minutes.

The faradaic current should be carefully and thoroughly applied to every atrophied muscle within reach which responds, and should be powerful and slowly interrupted. In those muscles which do not contract to the induced current the primary may be employed, but such a course will rarely be necessary, the muscle being, in the vast majority of cases, beyond the reach of remedial means. It is probably entirely atrophied.

By the use of these measures I have succeeded in curing three cases. These have already been referred to. The last, a man whose thenar and hypothenar eminences were markedly atrophied, and in whom the flexores carpi ulnaris and radialis were already affected, came with his physician to my clinic at the University Medical College. I advised the treatment mentioned; it was carried out, and in the course of two months the muscles were almost completely restored. The atrophy showed no further disposition to extend. I have since heard that this patient entirely recovered.

If there is the least suspicion of syphilis, iodide of potassium in large doses should be administered. In the case of a gentleman affected with progressive muscular atrophy, with an undoubted clinical history of syphilis, and who, residing out of New York, I see only about once a month, a very positive arrest of the disease appears to have resulted from this treatment. When he first consulted me the right thenar and hypothenar eminences were entirely destroyed; the interossei and lumbricales were nearly so. All the muscles of the forearm were more or less affected, and the disease was manifesting itself in the left thenar eminence, which was already decidedly wasted. He was at first treated by electricity, but there was no improvement, and while this agent was being used the left triceps showed signs of atrophy, and fibrillary contractions occurred in the muscles of both arms, which were not yet wasted, and in those of the trunk. The electricity was now discontinued after having been employed over six weeks, and the iodide of potassium was administered in gradually-increasing doses, beginning with ten grains three times a day. At about the time thirty-grain doses were reached, the fibrillary contractions ceased. He continued to increase the doses till he took half an ounce a day. There were then no contractions, and no further extension of the atrophy had taken place. The medicine was now discontinued for ten days, when it was resumed and continued as before. He still takes the iodide in gradually-increasing doses every alternate month, up to forty grains three times a day. A year and more has now elapsed since I first saw this patient, and there has been no advance of the disease since the treatment with the iodide was begun, and no fibrillary contractions in any part of the body since their disappearance nearly a year since.

A few cases of improvement have been reported as occurring from hydro-therapeutics.

It is very probable that the majority of the instances in which ameliorations or cures are asserted to have been produced by one thing and another were not in reality cases of progressive muscular atrophy. Every physician, whose practice is extensive in the class of nervous diseases, has doubtless had many patients consult him in whom the diagnosis of progressive muscular atrophy has been made, but who were affected with very different affections from that very intractable malady.

b. Progressive Facial Atrophy.

The remarkable affection now to be described under the name of progressive facial atrophy has been known since 1825, when Parry[1] described the case to which all subsequently noticed have a more or less close resemblance. Although cases were subsequently reported it seems to have attracted little attention till Lande,[2] in 1869, and Frémy,[3] in 1872, published their monographs. No account of the disease has yet appeared in this country, and only one case has been reported in Great Britain since Parry's above cited. This case, described by Dr. Moore,[4] of Dublin, appears to have been a typical one, which is certainly not the fact with several of those quoted by Frémy.

The disease, which was called by Romberg—who was the first to give it an independent existence—trophoneurosis facialis, by Moore unilateral atrophy of the face, and by Lande laminar aplasia, does not seem to be very common. Eleven cases have been collected by Lande, and Frémy adduces twenty-four additional ones, several of which, however, are, as I have said, not cases of the disease in question. Three instances only have come under my observation.

Various theories relative to its essential character have been advanced. These, with the reasons which have induced me to consider it as having affinities with progressive muscular atrophy, will be fully brought forward under the head of morbid anatomy and pathology.

Symptoms.—The first case which occurred in my own experience was that of a lady forty-one years of age, who consulted me in January,

---

[1] Cited by Romberg, "Lehrbuch der Nervenkrankheiten des Menschen," Berlin, 1854.

[2] "Essai sur l'aplasie lamineuse progressive," Paris, 1868.

[3] "Étude critique de la trophonévrose faciale," Paris, 1872.

[4] "Case of Unilateral Atrophy of the Face," *Dublin Quarterly Journal of Medical Science*, 1852, p. 245.

1874. Twenty years previously she had noticed as the first symptom a very slight degree of weakness in those muscles of the left side of the face concerned in the movements of the lips, so that, when she attempted to smile or laugh, the mouth did not expand to the same extent on that side as on the right.

This condition lasted several months without giving her much annoyance, till on waking one morning she noticed a pale, almost white spot on the skin immediately over the left malar bone. This was of a sub-rotund form, and gradually enlarged to the size of a dollar, becoming paler in hue and more irregular in outline.

Then she began to notice that there was a lack of the fullness which characterized the right side of the face, and this was especially evident at the situation of the spot. Here a depression was plainly to be seen.

Then a second depression, but this time without being preceded by paleness of the skin, began to appear. This was situated at about the middle of the chin, half an inch to the left of the median line. This extended most toward the right side, and in the course of two years had reached the median line and had a length of about two inches toward the angle of the mouth.

During the time that these depressions were extending she had been subject to fibrillary contractions all over the left side of the face.

There were no other symptoms, beyond the exceedingly gradual extension of the first depression, for fifteen years. Then a third spot, situated on the skin immediately over the angle of the jaw, on the left side, appeared and gradually extended as had the first. A depression likewise occurred in the soft parts at this spot, and, extending, finally reached the first depression.

When she consulted me there was a marked difference in the size of the two sides of the face, especially the lower part. The skin over the forehead on the left side was glossy and the belly of the occipito-frontalis muscle was decidedly thinner than that of the opposite side. The left eye appeared to be less prominent than the right, the temporal muscle was thinner, and the masseter was certainly not so thick as its fellow. All the muscles of the angle of the mouth, as well as the left half of the orbicularis oris, were atrophied. The depression on the chin involved the depressors of the lower lip and angle of the mouth. The elevator of the upper lip and of the ala nasi was not affected.

The skin over the left side of the face was apparently attached firmly to the parts below, and did not admit of being moved or pinched between the fingers. It was decidedly thinner than that of the other side.

I could not ascertain that there was any atrophy of the bones. The pulsations of the carotid, temporal and facial arteries were as strong on the left side as on the right.

There was no discoloration or falling off of the hair, no aberration of sensibility, no unilateral sweating, and no difference in the amount of sebaceous secretions on the two sides.

The motor power of the left side of the face was weaker than that of the right. When the mouth was expanded, the action was markedly less on the left than on the right side. The left buccinator was thinner and weaker than the right, the left half of the orbicularis oris did not contract to the same extent as the right when the mouth was pursed up, and the jaws were less strongly brought together on the left than on the right side. Yet there was no paralysis in any muscle, and each, on very thorough exploration with the faradaic current of moderate power, contracted well.

Examined with the æsthesiometer the sensibility was found to be intact. At no time had there been numbness, pain, or any abnormal sensation.

The tears, saliva, and buccal and nasal mucus, did not appear to be altered, either in quality or quantity.

The tongue was not involved, and, when protruded, came out straight. Deglutition was unimpaired.

The temperature of the two sides of the face was examined by a delicate thermometer, but no difference could be found to exist; but in October, 1875, I again had the opportunity of examining this patient, and then, by means of Dr. Lombard's thermo-electric apparatus, I ascertained that the left was .7° centigrade lower in temperature than the right side. The general health was excellent.

Although not allowed to have a photograph taken, I obtained the permission of this lady to examine the muscular tissue, and puncturing the buccinator with Duchenne's trocar I succeeded, with some little difficulty, in extracting a fragment for microscopical investigation. For purposes of comparison, I operated in the same manner on the corresponding part of the opposite muscle. The results of the examination will be given when we come to the consideration of the morbid anatomy and pathology.

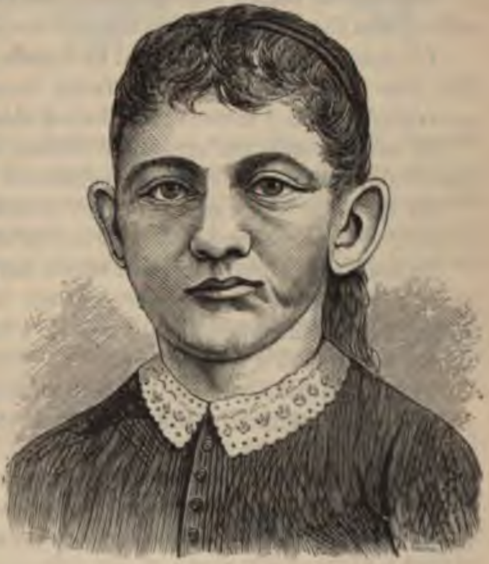
A second case came under my observation shortly after the publication of the foregoing in the sixth edition of this work, but the patient, a woman of about forty years of age, passed from my notice before I had the opportunity of making a study of the phenomena, or even of making notes of them. My recollection, however, is clear that the muscles supplied by the facial, the motor branch of the fifth, and the hypoglossal, were the seat of atrophy.

A third case[1] forms the subject of a communication read before the New York Neurological Society, March 2, 1880. The patient was a girl fourteen years old. The affection was of gradual growth, and did not attract marked attention till about two years previously to my seeing her. It was then noticed that the left side of the face was different from the right, and careful examination showed that there were two depressions: one just above the angle of the mouth, and one just below and a little external to the other. Subsequently, the one above and slightly in front of the left ear began to appear. All of them have continued to increase up to the present time (January 24, 1881), and in addition there is a decided difference in the size of the two sides of the face (Fig. 64). There has at no time been any apparent paralysis. Occasionally, there are what may be called paroxysms of numbness, extending over the left side of the face and never passing the mesial line. These only last a few minutes. At no one of my examinations have I been able to detect any loss of sensibility except of a limited region over the left half of the orbicularis oris muscle. The centres of atrophy were not preceded by any whiteness of the skin. The hair, however, is markedly thinner on the antero-superior auricular centre of atrophy than on the sound side.

---

[1] "A Case of Progressive Facial Atrophy, with Remarks on the Pathology of the Disease," *Journal of Nervous and Mental Diseases*, April, 1880.

Fig. 64.



Examination shows, what had not previously been noticed, that the left half of the tongue is much smaller than the right, and that the palatine arch on the same side is flatter than on the opposite side. The tongue when protruded is deflected toward the affected side. There is no difficulty of swallowing, no defective articulation, no loss of taste, and no deficient sensibility of the tongue or any part of the mucous membrane lining the buccal cavity.

The first symptom which ordinarily makes its appearance is the white spot, which shows an evident tendency to extend. The centre of greatest atrophy is in intimate topographical relation with this spot, and it is here, therefore, that the depression is most marked.

The skin becomes thinner, as is well perceived when a fold of it is pinched between the fingers, as can be done in the early stage of the disease. The cellular tissue also diminishes in volume.

The hair, eyebrows, eyelashes, and beard, generally either fall out or lose their color, changing to a gray or even perfectly white hue. The sebaceous secretion is usually less on the affected than on the sound side. Sometimes the larger arteries are apparently diminished in calibre, but the capillary circulation, as evidenced in blushing, is as active on the affected side as on the other.

The muscles have generally been atrophied both in thickness and length. Fibrillary contractions have sometimes been observed. It is probable they would be generally noticed if attention were directed to them.

It is rarely the case that sensibility is disturbed; but occasionally neuralgic pains have been experienced. The cartilages and even the bones have been sometimes the seat of atrophy.

The special senses remain intact, and the secretions of the tears, the saliva, and the buccal mucus, are not diminished.

Of the eleven cases collected by Lande, the tongue was atrophied on the side corresponding to the facial disease in five cases, and, when protruded, pointed toward the affected side.

In several cases the atrophy extended to the veil of the palate and the uvula; but the function of deglutition has never been impaired.

In three of the cases cited by Lande, the atrophy affected the larynx. Phonation was impaired in one of these instances.

In none of Lande's cases in which the point was inquired into was there any difference in the temperature of the two sides. In five of Frémy's cases the affected side was of a temperature lower from a few tenths to one and a half degree.

In no case has there been complete paralysis of any muscle, and the portion which remains, always contracts to the excitation of the electrical stimulus.

Frémy's statistics are very much to the same effect as those of Lande, though they are, I think, open to the objection that some of his cases were not true instances of the disease. Of twenty-seven cases cited by him, of which details are given, the tongue was affected eight times, the lips nine, and the veil of the palate five times. In seven other cases no statement is made in regard to these points, and in one it is vaguely stated that there was buccal atrophy. In four of these cases the affection involved at the same time both lips, the tongue, the veil of the palate, and its pillars on one side.

The progress of the disease is exceedingly slow, the condition existing in many cases for several years. It appears, however, to be distinctly progressive in character. No death has occurred from it, nor has any post-mortem examination been made with the view of inquiring into the nature of the affection in any patient dying of an intercurrent disease.

The accompanying figures from Lande represent the face of a woman affected with progressive facial atrophy. In Fig. 65 a front view of the countenance is given, and the atrophy of the left side is clearly shown. Fig. 66 represents the left side of the face; and, for purposes of comparison, the right, unaffected side, is given in Fig. 67.

FIG. 66.

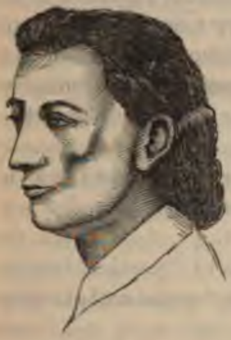


FIG. 65.

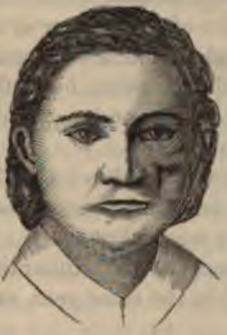
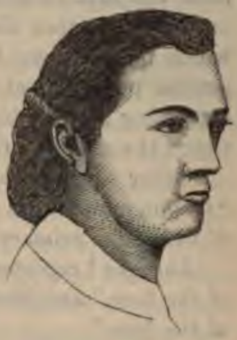


FIG. 67.



Causes.—Little is known relative to the etiology of this singular disease. It appears, however, generally to originate during early or adult life, and females are more subject to it than males. In one case it ensued after a fall on the head, and in one it followed an attack of scarlet fever. No evidence of hereditary transmission has been adduced.

Diagnosis.—Lande gives a long list of diseases from which facial atrophy is to be diagnosticated. I do not see that the affection is likely to be confounded with any other than progressive muscular atrophy, and, perhaps, in some cases, in its early stages with facial paralysis.

As regards the first of these—progressive muscular atrophy—it rarely if ever begins in the face, and is not confined to that part of the body in any case. Moreover, there is discoloration of the skin, and no cutaneous atrophy. Instead of being tightly stretched over the soft parts below, the skin is loose and can be easily taken up in a fold between the fingers. When the face is its seat, as it sometimes is, secondarily, its manifestations are not confined, as are those of facial atrophy heretofore observed, to one side. The lesions as regards the face, the tongue, and deglutition and phonation, are much more profound in progressive muscular atrophy than in facial atrophy.

Relative to facial paralysis (Bell's) there can ordinarily be no difficulty in making a diagnosis. As in my case, there may be a marked weakness of the facial muscles in the first stage of the disease under notice. But the mode of origin—Bell's paralysis coming on suddenly—and the fact that in it the electric contractility of the muscles is always diminished, while in facial atrophy it is unimpaired, will suffice for the distinction.

Prognosis.—No case of a cure is on record. The affection is not one which, as heretofore observed, terminates in death, but it is evident that there are cases in which it shows a tendency to involve organs of which the perfect integrity is essential to life.

Morbid Anatomy and Pathology.—Bergson,[1] who appears to have been the first to study the disease under consideration, regarded it as not due to either disorder of the motor or sensory nerves or of those which preside over the glandular secretions. Without indicating the precise primary seat of the affection, he looked upon it as essentially consisting in a morbid state of the layer of cellular tissue situated between the skin and the muscles.

Other cases were reported, and in 1851 Romberg[2] described it as a "tropho-neurosis of the face," a disease characterized by atrophy but of which the primary seat was unknown.

Lasegue[3] reported a case in 1852 under the title of "partial atrophy of the face," and Moore,[4] in the same year, called it "unilateral atrophy of the face."

None of these writers made any decided effort to locate the disease or to interpret its real nature till, in 1860, Samuel,[5] citing a well-marked case, first reported in 1848 by Hueter, advanced the opinion that progressive facial atrophy was an affection of the trophic system of nerves, and, following Moore, he designated it unilateral atrophy of the face.

Then, as we have seen, Lande,[6] in 1869, wrote a very complete monograph on the disease, which he called "laminar aplasia" (aplasie lamineuse), by which term he intended to convey the idea which he entertained of its nature, that it was an affection of the cellular tissue primarily.

Subsequently, Eulenburg[7] very fully described the malady under the name of "hemiatrophia facialis progressiva," and, taking into consideration the fact that the manifestations of the disease are exhibited in those parts which are supplied by the fifth pair of nerves, he regarded it as the result of a lesion of this system, or at least of a derangement of its function.

Finally, Frémy,[8] in a monograph of great excellence, enters at length into a consideration of the pathology of the disease, and concludes that it is to be classed with those trophic neurotic disorders which have been studied by Romberg, Samuel, Charcot, and Vulpian, and that it essentially depends upon derangement of the trifacial nerve.

---

[1] "De Prosopodysmorphia sive nova Atrophia facialis," Berlin, 1837, cited by Lande.

[2] "Klinische Wahrnehmungen und Beobachtungen," Berlin, 1851.

[3] "Atrophie partielle de la face," *Archives Générales*, tome xxix., 1852.

[4] "Case of Unilateral Atrophy of the Face," *Dublin Quarterly Journal of Medical Science*, 1852.

[5] "Die tropischen Nerven," Leipzig, 1860.

[6] "Essai sur l'aplasie lamineuse progressive (atrophie du tissue connectif) celle de la face en particulier," Paris, 1869.

[7] "Lehrbuch der functionellen Nervenkrankheiten," Berlin, 1871.

[8] "Étude critique de la trophonevrose faciale (Physiologie pathologique)," Paris, 1872.

All these opinions have been thoroughly considered by Vulpian.[1] He shows very conclusively that progressive facial atrophy is not a disease of the sympathetic system, and then, in further illustration of his views, says:

"Certain peculiarities of this affection seem to indicate that the trophic disorder of the face is produced by an intracranial lesion. But the difficulties are so great in the way of imagining that a limited lesion could give rise to all the alterations which occur in the face, the hair, the buccal cavity, and even in the neck, as in some cases, that we can see how M. Lande was led to reject the idea of a primitive lesion of the nervous system, and to admit only a protopathic lesion of the cellular tissue of the face. At the same time I do not think that his doctrine will obtain many partisans. Indeed, it is very difficult to abandon the idea of an intracranial lesion as the cause of the trophoneurosis. This affection is produced in a certain number of cases as a consequence of traumatic violence inflicted on the head or face. Its development is accompanied, in the great majority of cases for several years, with pains of greater or less violence seated in the head, ordinarily toward the fronto-temporal region. Sometimes there are spasmodic movements of the muscles of the face or of the jaws. In some rare cases there has been numbness in the superior extremity of the opposite side. These are the circumstances which seem to point to a cerebral lesion. But we cannot affirm that such lesions exist, while we have no post-mortem examination to enlighten us on this point, and while we are embarrassed to designate a seat for the lesion, which can reasonably explain all the phenomena of the disease. It has been proposed to attribute the trophoneurosis to a lesion of the ganglion of Gasser, but can we cite a single case in which lesions of this organ have existed in conjunction with an *ensemble* of symptoms such as that presented by the disease under notice? If it be true that the greater part of the alterations produced in the malady are in the region supplied by the trigeminus, and even in the course of certain of its branches (cicatricial depression of the forehead in the course of the frontal branch), we are compelled to admit that it is not so with all the changes (for example, those of the neck, rare, it is true). The special atrophy which is shown in the affected regions is not easily explained in the present state of our knowledge by the modifications of nutrition resulting from lesions of the trigeminus. We see nothing similar to the lesions of facial trophoneurosis produced as a consequence of experiments made on this nerve or on the ganglion of Gasser. We ought not to forget, however, the nutritive troubles of the cornea, so common in lesions of the ganglion of Gasser and rare in facial trophoneurosis. Then in some cases there is atrophy of certain facial muscles, whatever M. Lande may say to the contrary; and we do not know, either clinically or by experimentation, that muscular atrophy is ever directly produced by alterations of the trigeminus or its ganglion. For to speak only of the tongue, the lateral half of which is so often atrophied in facial trophoneurosis, I have demonstrated that section of the lingual nerve is not followed by appreciable atrophy of the lingual muscles.

---

[1] "Leçons sur l'appareil vaso-moteur," tome ii., 1875, p. 432.

"The difficulties which we encounter, when we attempt to connect the trophoneurosis with a lesion of the trigeminus, are increased, when we seek to explain the production of this disease by an encephalic lesion seated, for example, in the vicinity of the nucleus of origin of the fifth pair.

"To conceive an hypothesis so little plausible, we would be forced to suppose the existence of multiple lesions seated in one of the halves of the isthmus of the encephalon. But all tentative explanation appears to me to be perfectly vain, since we are ignorant whether there is or is not a primary lesion of the nerves or the nerve-centres. We can, however, positively affirm that, taking into consideration all the characteristics of facial trophoneurosis, it is not due to vaso-motor perturbation acting on the parts which are the seat of the disease."

As we have seen, there has been thus far no examination of the nerve-centres, the nerves, or the muscles. But, in the case which was under my observation, I obtained, as stated, portions of the sound and atrophied buccinator muscles, and submitted them to careful microscopical examination. The result was that I ascertained that the fibrillæ of the atrophied muscle exhibited no evidence whatever of degenerative changes—the transverse and longitudinal striæ were distinct, and there were no traces of fatty degradation. But the transverse diameter was reduced to about one-third the normal size, as is seen in the cuts herewith given, which are drawn from the camera lucida to an exact and uniform scale when magnified four hundred diameters. In Fig. 68 is shown a single fibre from the right buccinator muscle, and in Fig. 69 three fibres from the corresponding part of the left buccinator. Fig. 70 represents a transverse section of the right, and Fig. 71 one of the left buccinator muscles.

Examination also shows that not only is the diameter of the fibrillæ markedly diminished, but the length is also lessened, as is evidenced by the fact that the transverse striæ are very much closer together in the atrophied than in the sound fibrillæ.

It will likewise be perceived that there is in the affected muscle a notable diminution of the thickness of the layers of the internal perimysium, or connective tissue, which separates the fibres from each other. This tissue appears to be somewhat hypertrophied on the right side.

This, therefore, constitutes the first positive contribution to the morbid anatomy of progressive facial atrophy, but, small as it is, it affords very important indications in regard to the nature and seat of the affection.

In the third case, the one of which the photograph has been given, I obtained by like means portions of the muscular fibre from each side, and they were exhibited to the Neurological Society. They were taken from the buccinators. In the one from the right or normal muscle, the primitive bundles are seen to be of full size and in every respect of healthy appearance. In the left or affected muscle, the bundles are perceived to be less than one third the diameter of the others, and to be much paler in hue. There is no trace of fatty degeneration, not a single fat-corpuscle or oil-globule being visible anywhere. The difference is so striking, that one can scarcely resist the at least momentary belief that a sudden change in the magnifying power has been made. Accurate measurement shows that the bundles of fibres from the sound muscle are of the average diameter of $\frac{1}{650}$ of an inch, while those from the unsound muscle are only $\frac{1}{2200}$ of an inch. The size of the fibres from the sound side is, therefore, greater than that ordinarily existing in the facial muscles, and may probably be indicative of hypertrophy.

FIG. 68.

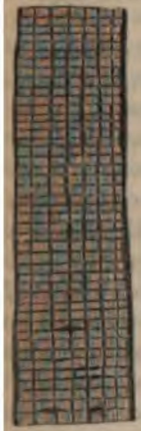


FIG. 69.

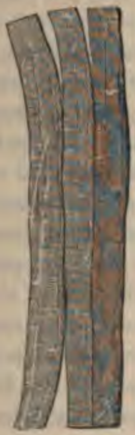


FIG. 70.

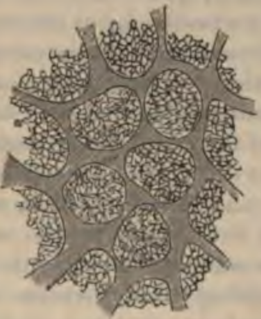
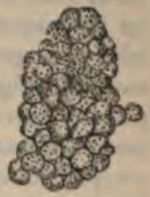


FIG. 71.



Thus, in two cases in which microscopical examination has been made of the muscular tissue in progressive facial atrophy, there has been found an identity of lesions—atrophy without degeneration. I hence feel warranted in concluding, at least till these results are successfully controverted, that this is one of the concomitants of the disease.

It shows that progressive facial atrophy is not one of those diseases manifested by degenerative changes of the muscles such as we have seen take place in infantile paralysis, spinal paralysis of adults, pseudo-hypertrophic paralysis, and progressive muscular atrophy. It is an atrophy pure and simple, without the slightest tendency to degeneration.

So far as analogy is concerned, there is a marked affinity, not to say resemblance, between the symptoms of progressive muscular atrophy affecting the muscles of the face, the tongue, and the pharynx, and those of some cases of progressive facial atrophy, in which not only the face is involved, but also the tongue, and in one case at least the larynx. We have seen that in glosso-labio-laryngeal paralysis the muscles of the same regions are involved, but instead of atrophy we have paralysis. Now, when we come to seek out the primary seat of progressive muscular atrophy affecting the face, tongue, and throat, and that of glosso-labio-laryngeal paralysis, we find both in the bulb and especially in the nuclei of origin of the facial, the hypoglossal, the spinal accessory, and pneumogastric nerves. If two such different but cognate diseases may occupy the same anatomical situation, why may not progressive facial atrophy, different but cognate, be also an affection of the same region? The fact that the atrophy involves other parts than the muscles, is no valid objection against this hypothesis. We have seen that in infantile spinal paralysis there is sometimes an atrophy of the bones. And yet we all agree to consider this disease as a primary affection of certain cells in the anterior tract of gray matter.

The examination of this case, as of the two others I have witnessed, shows that muscles supplied by the motor branch of the fifth nerve, by the facial and by the hypoglossal, are atrophied; that the skin, hair-bulbs, cellular tissue, and even the bone (temporal), are similarly affected; and that there are sensory disturbances in the skin supplied by the fifth nerve. Under these circumstances I arrive at the conclusion that the nuclei of these nerves are the primary seat of the disease in this case.

The only other view that it appears necessary to discuss in this connection is the one that all the phenomena may be the result of primary implication of the fifth nerve or its nuclei. The involvement of the motor nucleus only would certainly not account for the multiple muscle lesions observed in this case; the only muscle affected supplied by the motor branch of the fifth nerve is the temporal, and this only in a very limited portion of its substance. We have, therefore, merely to inquire as to the implication of the sensory nucleus, it being admitted that the motor nucleus is to some extent affected, as shown by the effect produced upon the temporal muscle. The existence of a third root, as contended for by Merkel,[1] and to which he assigns trophic functions, can scarcely be regarded as demonstrated; and, though its probability may be admitted, we need not in the present state of our knowledge take its possible influence into consideration. So far as the derangements of sensibility are concerned, it is conceded that they are due to lesion of the sensory nucleus or of the nerve itself in some part of its course.

---

[1] "Die trophischen Wurzel der Trigeminus," *Centralblatt*, 1874, p 902.

Now, how far could a lesion of the nucleus of the sensory root of the fifth nerve, or one of the root itself, tend to produce all the phenomena observed in this case and others of progressive facial atrophy?

If the intra-cranial portion of the nerve be divided, we meet, in addition to loss of sensibility in the parts to which the nerve is distributed, with an invariable series of results which are entirely different from those observed in progressive facial atrophy. These, however, are intimately related to the function of nutrition. Thus, the cornea ulcerates, the conjunctiva becomes inflamed, the glands innervated by the nerve have their functional activity diminished or altogether arrested, and occasionally, apparently by reflex influence, ecchymoses appear in the lungs and stomach.

Certainly these are not the accompaniments of progressive facial atrophy.

The phenomena due to an irritation of the sensory nucleus, or of the nerve in any part of its course, are so entirely different from those characterizing the disease in question, that it is not necessary to dwell upon them more particularly.

It appears to me, therefore, that all the atrophic phenomena present in cases of progressive facial atrophy are, like those met with in progressive muscular atrophy and spinal paralysis of infants and adults, the result of lesion of the nuclei of motor nerves—and probably of trophic cells—forming with the motor cells the centres of origin of these nerves. In these diseases atrophy takes place without the intervention of any sensory nerve or sensory root, and there is, therefore, no necessity for the introduction of the sensory part of the trigeminus into the pathological circle presiding over progressive facial atrophy.

So far as the motor nerves which are in relation with the parts affected in progressive facial atrophy are concerned, we know very well that, in other diseases in which their functions are abolished wholly or in part, the resulting paralysis is always accompanied with atrophy—the nerves, of course, containing the fibres coming both from the trophic and motor cells of the nuclei. Take, for instance, the hypoglossal, a purely motor nerve. There are a few cases on record in which the hypoglossal, on one or both sides, has been so compressed by tumors that its functions were completely interrupted, and this interruption was invariably followed in a short time by atrophy. Lockhart Clarke divided one of the hypoglossal nerves in a rabbit, and within a month after the operation the corresponding half of the tongue was markedly atrophied.

It may be well to allude to the theory that progressive facial atrophy is the result of lesion of the sympathetic system—if only to say that there are no facts which tend to its support.

In an interesting paper based upon two cases, Dr. Bannister[1] arrives at the conclusions that the trophic functions of the fifth nerve are especially implicated, and that in some cases there are positive lesions of other cranial nerves. He considers it proved that the symptoms indicate a chronic trophic asthenia or paralysis rather than any irritative action.

I am, therefore, of the opinion that progressive facial atrophy is an affection of the trophic cells of the bulb which are the nuclei of the facial, the hypoglossal, and the spinal accessory nerves; that ordinarily the lesion does not extend farther than the facial, but that sometimes when the tongue is involved it reaches the nucleus of the hypoglossal and occasionally that of the spinal accessory. In these cases in which there are aberrations of sensibility the nucleus of the sensory root of the fifth pair may be affected, and in those in which the temporal and masseter muscles are involved the motor root may also be implicated. Or the pain which is sometimes an accompaniment of the disease may be due to the contracting process going on in the muscles and connective tissue by which the terminal branches of the trigeminus are compressed.

Why the atrophy should so generally affect the left side of the face in preference to the right, I do not pretend to explain; but, since the recognition of aphasia and its association in the vast majority of cases with lesions of a circumscribed region of the left hemisphere, we need not be surprised at the additional instance of hemitopology, incomplete as it is, afforded by progressive facial atrophy.

Finally, the question may be asked, Why should the manifestations be restricted to one side? I should answer that I do not know, any more than I am aware why ptosis or external strabismus should affect the eyelid and eyeball of one side; or why hemi-chorea should exist; or why, when a person has an attack of cerebral hæmorrhage, he should not straightway have another on the opposite side of his brain.

The first two cases reported occurred on the left side, then there was one on the right, and then eight on the left. If the third case had escaped observation, we should have had to appearance a uniform implication of the left side, to the exclusion of the right. Now about a dozen cases are reported as involving the right side.

It appears to me, however, that the indisposition manifested to pass the mesial line is a strong argument against the affection being a local lesion only.

Treatment.—Slight success was obtained by Hueter and Moore by the use of faradaic currents to the atrophied region. I employed both these and the primary current, the latter to the nucha as well, in two of the cases under my care, but without perceptible effect. I also administered strychnia and other tonics without benefit. This treatment, however, seems to be indicated, and is in general urged by those who have written on the subject. No cure has yet been reported. It must be borne in mind that diseases which are slow to advance are also slow to recede.

---

[1] "Progressive Facial Hemi-atrophy," *Journal of Nervous and Mental Diseases*, October, 1876.

III.

INFLAMMATION LIMITED TO THE POSTERIOR TRACT OF GRAY MATTER OF THE SPINAL CORD.

The functions of the posterior tract of gray matter being, so far as we know, exclusively sensory, we should expect to find inflammation of this region manifested exclusively by aberrations of sensibility. So far, however, nothing has been done toward the recognition of the symptoms of such a lesion, and the localization of their immediate cause in the posterior horns of gray matter. According to Charcot, we only know that when they are the seat of a profound alteration there is more or less pronounced cutaneous anæsthesia of the corresponding side of the body.

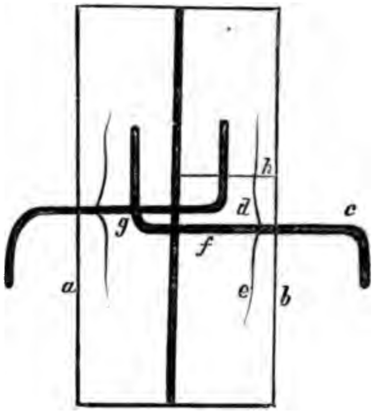
The posterior tract of gray matter is certainly an important part of the channel by which sensory impressions reach the brain from the parts below. It is quite certain, not only from the teachings of experimental physiology, but also from the instruction we derive from disease and injury of this region, that when it is profoundly altered there is a corresponding degree of anæsthesia in the parts below; not, however, according to my experience and that of others, on the same side as the lesion, but on the opposite side. To Brown-Séquard we are mainly indebted for a knowledge of the fact that the gray matter of the cord is the conducting medium for sensory impressions; that there is a decussation of the conductors is also a fact which he established.

If, therefore, a lateral half of the spinal cord be divided so as to include the whole of the gray matter, the animal upon which the experiment is performed loses sensibility in the parts below, on the opposite side of the body, and—which is not, however, a matter of present inquiry—motion on the same side.

Cases in which these phenomena—loss of motion on one side and of sensibility on the other—are coexistent from spinal disease, are by no means very infrequent. Several such have been under my care in hospital and private practice, and I have always attributed them to a lesion of one lateral half of the cord disturbing the power of motion on the same side and of sensation on the other.

But experiment shows that, while one part of the posterior nerve-roots passes over to the opposite side immediately on its entrance into the cord, another part passes upward and another downward. The effect, therefore, of a limited lesion involving one lateral half of the cord would be profound anæsthesia of the opposite side of the body and a slight degree on the same side. Accordingly in such cases as those I have referred to, there is always a trace of numbness on the side the motion of which is paralyzed. The action of a lesion of one lateral half of the cord in only slightly diminishing sensibility on the side of the alteration while greatly lessening it on the opposite side will be readily understood from an examination of the accompanying diagram, Fig. 72; *a*, the left half of the spinal cord, *b* the right half; *c*, a right posterior root, with its ascending fibres *d*, its descending *e*, and its decussating fibres *f*; *g* decussating fibres from the opposite side. A lesion of the right side of the cord at *h* will produce great loss of sensibility on the opposite side, and slight loss on the same side. That the decussation takes place in the gray substance has not been absolutely settled by anatomical demonstration, but it remains extremely probable, mainly by the fact that the commissure is composed chiefly of gray tissue.

FIG. 72.



Dalton[1] says in relation to this matter: "The transmission of sensitive impressions, therefore, takes place through the gray matter. This substance, which is itself insensible to direct irritation, forms the medium of communication between the peripheral fibres of the sensitive nerves and the brain above."

And again:

"It is certain, however, that after section of one lateral half of the cord the phenomena which indicate a crossing of the sensitive tracts are distinctly marked. We have repeated this experiment, and have found that after such a section in the dog, in the dorso-lumbar region, the difference in the effects produced upon sensation and motion on the two sides is very striking: sensibility is either lost or very much diminished upon the opposite side, while upon the same side with the section, where there is complete muscular paralysis, the sensibility remains and is increased in intensity.

---

[1] "A Treatise on Human Physiology," sixth edition, Philadelphia, 1875, pp. 353-356.

With this brief statement of the physiology and pathology of the subject as at present known, I leave the further discussion of the diseases of the posterior tract of gray matter till science has given us more definite information than we now possess relative to its functions and derangements.

IV.

INFLAMMATION OF THE ANTERIOR AND POSTERIOR TRACTS OF GRAY MATTER OF THE SPINAL CORD.

The gray matter of the spinal cord as a whole, is subject to at least one disease—tetanus—which, according to recent investigations, is in reality a central myelitis. Since Lockhart Clarke in 1864 gave the results of his examinations on this subject, other data to a like effect have been published, and, though differences in the lesions have been observed these are of secondary importance to the main fact that in tetanus the central gray matter is the chief seat of the alterations. The circumstance that the white matter has also been found diseased no more invalidates the correctness of the statement than the fact that a patient dying with the symptoms of pneumonia, still has that disease, even though there be a patch or two of inflamed pleura as a secondary lesion.

a. Tetanus.

Two varieties of tetanus are generally described by systematic writers—the idiopathic and the traumatic; but, as they are characterized by similar phenomena, differing mainly as to their modes of origination and severity of their symptoms, there would be no advantage in considering them separately.

Symptoms.—The first symptom to make its appearance in cases of tetanus is a feeling of pain or oppression in the epigastric region. In the beginning it does not attract much attention, but, as the disease advances, it becomes exceedingly severe, and adds greatly to the discomfort of the patient.

Soon after the occurrence of this pain, uneasiness is generally observed about the throat. This is, perhaps, no more than a sense of stiffness of the muscles concerned in deglutition, but it is not long before swallowing is impeded to a considerable extent. With these symptoms there are ordinarily mental and physical depression, sensations of chilliness, and a general feeling of *malaise*.

The foregoing constitute a prodromatic or formative stage, which may last a few hours or several days, and which is occasionally overlooked when the disease is intense and rapid in character.

In the next stage the epigastric pain is still a prominent symptom.

It is seated just below the sternum, and generally extends backward to the spinal column. It appears to be due to spasm of the diaphragm, so that this muscle is among the first, if not the very first, to be affected in the vast majority of cases. The difficulty of swallowing increases, and then the muscles of the jaws become contracted, constituting the condition known as trismus or lockjaw. At first there is only stiffness of these muscles with those of the neck, but gradually they become rigid, and the patient experiences difficulty, if not impossibility, in opening the mouth. The facial muscles do not escape, and an expression like the risus sardonicus is produced from the retraction of the angles of the mouth, the elevation of the alæ nasi, and the expansion of the nostrils. At the same time the eyes are staring, the brows corrugated, and the countenance anxious or wearied in appearance.

Sometimes gradually, at others suddenly, the morbid action extends to other muscles. Generally it passes to those of the neck, the back, and the loins, causing violent contraction, and bending the body backward. This state is called opisthotonos. The contraction of the powerful muscles referred to is so great as to cause the body to assume the form of an arch, the head being thrown far back, the abdomen protruded, and thus, if the patient were placed on his back, only the occiput and heels would touch the bed. Opisthotonos is the usual variety of spasm.

Two other forms are occasionally met with. In one of these—emprosthotonos—the body is bent forward from the contraction of the thoracic, abdominal, and pelvic muscles. In the other—pleurosthotonos—it is bent laterally. This latter may be met with in opisthotonos, owing to the muscles on one side being more strongly affected than on the other. Both emprosthotonos and pleurosthotonos are rare. Of very many cases of tetanus that have been under my observation, I have only seen the former four and the latter three times. The spasms characteristic of the disease are tonic; but, though they do not entirely relax, they are marked by more or less exacerbation, according to the severity of the attack, and the care taken of the patient. Any cause calculated to excite reflex action will induce an accession. Thus the contact of the bedclothes with the body—the legs especially—the touch of the hand, the forcible shutting of a door, the rumbling of carriages in the street, even the blowing of a breath of air on the skin, may produce an aggravation of the spasm. Even without any apparent excitation these fits occur. They are marked by great pain, and may be so violent as to break the teeth, and the bones of the legs, and tear the large muscles of the thighs. During their continuance, and often when they are not present, the pain at the pit of the stomach becomes unendurable, and the patient may lose consciousness through its intensity. I have several times seen this event occur.

The tonic rigidity of the muscles of respiration induces difficulty of breathing, and the same result may ensue from spasmodic closure of the glottis. Death has frequently taken place suddenly from one or other of these causes. With all this muscular excitement and mental disturbance there is in the early stages rarely any fever. Toward the close, however, the skin is hot, and the thermometer often ranges from 105° to 110° Fahr., or even higher, but the pulse remains small and weak.

Owing to the difficulty of swallowing, the patient suffers from hunger and thirst, and thus the powers of the system are still further reduced. The bowels are always obstinately constipated.

Wakefulness is generally present from the first. When the patient does sleep, it usually happens that the muscles are relaxed, to be again suddenly affected with spasm as soon as he awakes.

The mind is clear throughout, even in the most severe cases. When loss of consciousness occurs from extreme pain, it is from syncope, and not from any implication of the brain in the essential nature of the disease. Death usually takes place by apnœa. It may, however, result from exhaustion, and, according to some authorities, from the spasmodic action attacking the heart.

The duration of the disease is very variable. The shortest case on record is one observed by Prof. Robinson, of Edinburgh. The patient, a negro waiter, cut his finger with a piece of broken china. He was immediately seized with tetanus, and died within fifteen minutes. Mr. Poland quotes a case in which death took place in five hours; in a case cited by Lepelletier in a few hours; in one by Dr. Jackson in twelve; in one by Dr. Leith in eighteen; and in one observed by Mr. Curling in nineteen.[1]

The shortest duration in any case I have witnessed was twenty-six hours, though I believe there were several much shorter, which occurred during the recent war in this country.

The average period of duration in fatal cases is from the third to the fifth day. Instances in which it has been prolonged far beyond this limit are not uncommon. Hennen[2] reports a case in which it lasted six weeks, and then the patient died of another disease. He reports another case in which it lasted seven weeks, and ended in recovery. I have seen three cases in which it extended to the fifth week.

The period which elapses between the reception of the cause and the beginning of the symptoms is also subject to great variation. In a case already cited it was only fifteen minutes; in another, quoted from Dr. Randolph by Reeves,[3] the spasms ensued immediately after the patient was stung by a bee; and in another, which occurred in his own experience, they came on in a sensitive female immediately after running a needle into her finger. There is doubt, however, as to such cases really being tetanus. In the last one cited it is stated that "the body and extremities were rigid, mouth closed, and the jaws fixed, the eyes the same. At short intervals the whole body was affected with convulsive shocks; the administration of a dose of chloroform removed them, but the back and neck remained rigid for three days." This attack was probably a manifestation of hysteria. In eighty-one cases collected by Mr. Curling, the disease began between the fourth and fourteenth days, both inclusive, and in nineteen on the tenth day. The following table from Reeves shows the period of the occurrence of the disease in three hundred and forty-three cases:

---

[1] All the above instances are quoted from Reeves's "Diseases of the Spinal Cord and its Membranes," London, 1858, p. 387, *et seq.*

[2] "Observations on some Important Points in the Practice of Military Surgery," etc., Edinburgh, 1818, p. 263.

[3] *Op. cit.*, p. 377.

Within six, twelve, eighteen, or twenty-four hours	12
From 1 to 2 days	12
" 3 " 5 "	37
" 6 " 8 "	94
" 9 " 12 "	77
" 12 " 14 "	52
" 15 " 17 "	25
" 18 " 20 "	9
" 21 " 23 "	9
" 24 " 26 "	6
" 27 " 29 "	9
" 30 " 32 "	1
	343

Causes.—The most common cause of tetanus is bodily injury of any kind, from the slightest to the most severe, and of any part of the body, although wounds of some parts, as of the thumb and great-toe, are more apt to be followed by the disease than those of other regions. It has been known to result from the bite of a tame sparrow, from the sticking of a small fish-bone in the pharynx, from a seton in the thorax, from the stroke of a cane across the back of the neck, from the blow of a whip-lash, from fractured bones, and from every other imaginable wound or injury. In a case under my charge in this city, it was caused by a splinter of wood slightly scratching the palm of the hand; in another a slight punctured wound of the foot produced it.

Next in frequency to wounds, tetanus is induced by exposure to cold and damp. This is the exciting cause in the great majority of cases of idiopathic tetanus, and it increases liability in those who have suffered from wounds. It was not uncommon, during the recent war, for the number of cases of tetanus to be very much increased immediately after a sudden change of the weather from dry and mild to wet and cold.

It has also apparently been caused by worms, by abortion and labor, and by diseases of the womb. Terror has the reputation of having in-ed tetanus in one case reported by Dr. Willan, and in others ob- ved by Hennen.

In the form occurring in very young children, and known as tris- s nascentium, it appears to be induced by inattention to the cut um- cal cord.

The tendency to tetanus, especially among soldiers and others who e been wounded, is increased by poor diet, confinement in ill-venti- d hospitals, inattention to cleanliness, and neglect to give proper e to the wounds they may have received.

Diagnosis.—The only affections with which tetanus is liable to be founded, by any but the most ignorant, are the hysterical simulated ction, and the condition induced by poisoning with strychnia and er substances of its class.

That hysteria can simulate tetanus, as well as almost all other dis- es, we have abundant evidence. A case has already been referred in this chapter which was evidently hysterical, and several others e come under my observation. A lady now under my charge has eated attacks of hysterical spasms, during which her jaws are tight- losed, she is unable to swallow, and her body is bent backward so o assume the position of opisthotonos.

Such seizures are readily distinguished from tetanus by the facts t they are unaccompanied by pain or real distress, are of very tran- t duration, and are accompanied by other manifestations of hys- a.

From the artificial tetanus caused by strychnia, the diagnosis is re difficult; for, so far as the more obvious symptoms go, there is h a great similarity that even the most skillful diagnosticians might for a time, undecided. It is well known that strychnia is not unfre- ntly used for the purpose of committing murder or suicide, and it is sible so to employ it for either of these purposes as to cause its cts to extend over a long period of time, and thus to add to the iculties attending the discrimination. Even in such a case, however, diagnosis can be made if due care and a thorough inquiry into the tory of the case be made.

In the first place, the tetanus of strychnia always shows itself in lower extremities before trismus ensues. The legs are stretched lely apart, and the hands are generally involved. In natural tetanus, mus precedes spasm in the extremities; indeed, the lower extremi- are rarely affected to any great extent. The arms generally escape gether.

The epigastric pain, which constitutes so prominent a feature of e tetanus, is not present in the toxic variety. I have witnessed ee cases of poisoning by strychnia, and this pain was not complained in either of them.

In the tetanus of strychnia, the symptoms are developed with great rapidity, and death takes place generally within a half an hour, although life may be prolonged, in exceptional cases, somewhat beyond this period. In true tetanus it is very rarely the case that death takes place within twelve hours, and ordinarily not till several days have elapsed.

In those cases of poisoning by strychnia in which the doses have been small, and administered at comparatively long intervals, the symptoms are mitigated in violence, and consequently one of the distinguishing features of the two affections is lost. Still, the general character and sequence of the phenomenon are the same, and it is not improbable that careful observation and inquiry will fail to elicit the true nature of the case.

Prognosis.—The longer the time that has elapsed between the reception of the injury or subjection to other cause, the greater is the probability of a favorable termination. When the paroxysms are slight, and the intervals between them long, the prognosis is also more favorable. A low bodily temperature is a favorable indication. On the contrary, an elevated temperature is of fatal augury. The duration of the disease is likewise an important element in the prognosis; and, when it has lasted over a week, death does not often take place. Cases are, however, on record in which a fatal result has supervened after the affection has existed for several weeks.

Tetanus is, nevertheless, one of the most fatal of maladies. Dr. O'Beirne[1] witnessed two hundred cases without a single recovery. Hennen[2] never saw a case of acute symptomatic tetanus recover. McLeod[3] has collected and analyzed twenty-three cases which occurred in the British army in the Crimea, of which but two recovered. Demme[4] refers to eighty-six cases in the hospitals in Italy during the campaign of 1859, of which six were cured; and Hamilton[5] has observed eight cases, of which three recovered.

Nine cases have been under my immediate care, of which there were three recoveries. Of the many cases which I observed in the course of my inspections of camps and hospitals in the army during the recent war, I do not know how many terminated favorably. I am disposed, however, to believe that the number was not great. Hamilton states that his information leads him to think that, of one hundred and fifty cases which occurred during the war, the recoveries were few.

Morbid Anatomy and Pathology.—As regards the cord, the results of post-mortem examination of patients who have died of tetanus have up to a comparatively late period been very unsatisfactory. Rokitansky,[1] in chronic cases, found a proliferation of connective tissue. Wedl,[2] in one case, discovered increased redness of a portion of the spinal cord. Curling[3] declared that serous effusion with increased vascularity was generally observed in the membranes investing the medulla spinalis, and also a turgid state of the blood-vessels above the origin of the nerves; and Wunderlich[4] regarded the lesions as consisting in a proliferation of the connective tissue of the cord, the medulla oblongata, and the cornua cerebri and cerebelli.

---

[1] "Dublin Hospital Reports," vol. iii., pp. 343, 378.

[2] *Op. cit.*, p. 262.

[3] "Notes on the Surgery of the War in the Crimea," London, 1858, p. 153, *et seq.* Also table, p. 439.

[4] "Militär-chirurgische Studien," Würzburg, 1861.

[5] "A Treatise on Military Surgery and Hygiene," New York, 1866, p. 595.

But, in 1864, Dr. Lockhart Clarke,[5] after a careful examination of the spinal cords of six persons who had died of tetanus, found as the uniform results an abnormally enlarged condition of the blood-vessels throughout the gray matter, especially in the posterior horns, and granular disintegration of the nerve tissue. He expresses the opinion that tetanus depends (first) upon an excessively excitable state of the gray nerve tissue of the cord induced by the hyperæmia, and morbid condition of the blood-vessels, and the exudation and disintegration resulting therefrom, and (second) that the spasms are the result of the persistent irritation of the peripheral nerves by which the exalted excitability of the cord is aroused, and thus the cause which at first induced in the cord its morbid susceptibility to reflex action is subsequently the source of that irritation by which the reflex action is excited.

Subsequently, Dickinson[6] found enlargement of the blood-vessels throughout the gray substance of the cord, with perivascular exudation, rupture of the blood-vessels in many places, and granular disintegration.

Dr. Clifford Allbutt[7] has reported the results of his examination of the spinal cords in four cases of tetanus. He found diminution of the consistence of the cord of various degrees and situation; hæmorrhage in two cases visible to the naked eye; enlargement of the blood-vessels; exudation of a granular plasma surrounding the vessels; enlargement of the cells of the gray matter, and the granular degeneration of Clarke. Outside of this cord he found the nerve thickened and congested, and bathed in inflammatory products. These results were confirmed by the subsequent examination of Drs. Clark and Dickinson.

---

[1] "Beiträge zur Pathologie des Tetanus," *Virchow's Archiv*, tome xxvi., 1862.

[2] "Rudiments of Pathological Histology," "Sydenham Society Translation," London, 1855, p. 276.

[3] "A Treatise on Tetanus," etc., London, 1836.

[4] *Archiv der Heilkunde*, 1862.

[5] *Lancet*, 1864; *Medical Times and Gazette*, 1865; also, more fully, "On the Pathology of Tetanus," *Medico-Chirurgical Transactions*, vol. xlviii., 1865, p. 255.

[6] "Description of the Spinal Cord in a Case of Tetanus," *Medico-Chirurgical Transactions*, vol. li., 1868, p. 267.

[7] "On the Changes of the Spinal Cord in Tetanus," "Transactions of the Pathological Society of London," vol. xxii., 1871, p. 27.

Dr. Fox[1] made post-mortem examinations of four cases. In one the only abnormality remarked was dilatation and distention of the vessels of the spinal pia mater. In the others there were softening, hæmorrhage, amyloid bodies, in the gray substance, and thickening of the vessels.

Michaud[2] examined the cord in four cases. He found that the gray matter presented a general red appearance. The vessels were enormously enlarged. There were numerous free nuclei and foci of perivascular exudation. The gray substance, and especially the posterior commissure, was the seat of these alterations which, according to him, consist essentially in a proliferation of the nuclear elements of the connective tissue. The appearance which Lockhart Clarke considers to be a granular degeneration, Michaud regards as being due to these nuclei existing in the exudation around the blood-vessels. He considers tetanus to be an acute inflammation of the gray tissue of the cord.

When either of the upper extremities is the seat of the wound, which is the primary cause of the disease, the lesions of the cord are found in the cervical enlargement, and, when either of the lower limbs is injured so as to induce the affection in question, the spinal lesions are found in the lumbar enlargement.

The nerves coming from the wounded part have been found the seat of inflammation by Airlong and Tripier,[3] and by Michaud. In other cases they have not exhibited any change.

The muscles of the body suffer secondarily. The violent spasmodic contractions to which they are subjected often produce ruptures of their tissue and extravasations of blood.

On the other hand, it has often happened, especially in very rapid cases, that nothing has been found which could fairly be regarded as constituting the essential feature of the disease. Billroth[5] affirms that his examinations of the spine and nerves, in cases of tetanus, have thus far given only negative results, and this is in accordance with the observations of the great majority of pathologists. But these discrepancies are, I think, to be ascribed to defective methods of examination, and in no event can they disprove the positive data obtained by others.

It is contended by some authors that tetanus, like hydrophobia, is due to blood-poisoning. The fact, that a condition, so nearly resembling it as to be with difficulty diagnosticated from it, may be caused y the injection of strychnia into the blood, appears to favor this view. Iowever this may be, the character of the symptoms, as well as the natomical lesions, indicates the spinal cord to be the seat of the isease.

---

[1] "Recherches anatomo-pathologiques sur l'état des systèmes nerveux central et périphérique dans le tétanus traumatique," *Archives de Physiologie*, 1871, p. 59.

[2] "The Pathological Anatomy of the Nervous Centres," London, 1874, p. 355.

[3] *Archives de Physiologie*, 1870, p. 244.

[4] *Op. cit.*

[5] "General Surgical Pathology and Therapeutics, in Fifty Lectures," Hackley's translation, New York, D. Appleton & Co., 1871, p. 353.

The spinal cord is both an organ for the generation of nerve-force, nd for conducting impressions to and from the brain. In tetanus it is his first-named function which is deranged, and this is shown by the reat exaltation of reflex excitability which exists. Every thing capa-le of causing a reflex movement of the slightest kind, and even excita-ions which in health would be altogether unperceived by the cord, ugments its intrinsic action to a great extent where tetanus exists.

Now, we are able to produce a similar increase of reflex action by trychnia; and, in those cases of disease in which the amount of blood the cord is increased, very small quantities of strychnia produce the haracteristic phenomena of stiffness in certain muscles, and of aug-ented reflex excitability. The condition is aggravated by the medi-ine; and, if we had no other facts to support the theory, we should be arranted in concluding that, in cases of strychnia-poisoning, the mount of blood in the cord and the excitability of the organ are both ncreased. From a consideration of all the points bearing on the sub-ect, we are warranted in concluding that tetanus essentially consists n a morbid exaltation of the functions of the spinal cord as a nerve-entre.

Bernard[1] has investigated this matter with his usual exactness. He ays:

"Strychnia produces convulsions by exaggerating the sensibility of ertain parts; it also causes reflex movements. We have seen that the oint of departure is in the sensitive system; for, where the posterior oots of the nerves are cut, the animal dies without convulsions."

An experiment performed by myself and my friend and collaborator, Dr. S. Weir Mitchell,[2] shows that the action of strychnia is to destroy he nervous excitability from the centre to the periphery. Its influ-nce, therefore, must first be exerted on the spinal cord.

"Under the skin of a large frog, whose left sciatic nerve was pre-iously divided, a few drops of a strong solution of strychnia were in-roduced. Tetanic spasms ensued in two minutes. After forty-five inutes the nerves were irritated by galvanism. That of the left side, hich had been cut, responded energetically, while no motions could be roduced through the uncut nerve. The former remained excitable for wo hours later."

---

[1] "Leçons sur les effets des substances toxiques et médicamenteuses," Paris, 1857, . 386.

[2] "Experimental Researches relative to Corroval and Vao; Two New Varieties of oorara, the South American Arrow-Poison," *American Journal of the Medical Sciences*, uly, 1859; also "Physiological Memoirs," Philadelphia, 1863, p. 181, *et seq.*

Bernard[1] asserts that the action of strychnia extends no farther than the spinal cord; and any one who has seen a frog under the influence of this substance cannot have failed to notice that all the symptoms indicate exalted spinal action.

We are therefore led by observation and experiment to the conclusion that the lesion of tetanus is seated in the gray matter of the spinal cord, and that, although we cannot at present affirm an absolute identity of the lesions, in each case we have enough data to enable us to say in general terms that tetanus is essentially an inflammatory affection of the gray matter of the spinal cord.

Vulpian[2] has shown that strychnia does not produce organic lesions of the cord. He kept a frog for a month under its influence, and on killing the animal found the cord in all its parts in a perfect state of integrity. But on this point there is a difference of opinion, Jacubowitsch and Roudanowsky asserting that the processes of the nerve-cells are torn, and that the cells themselves are often ruptured. It is not, however, probable that the condition of the cord, in poisoning by strychnia, ever goes beyond the point of hyperæmia, which, being of recent occurrence, would disappear on death supervening. It is also extremely probable that, in the cases of tetanus in which recovery takes place, the organic derangements discovered by Lockhart Clarke do not occur. This is his opinion: Hyperæmia is the first stage of all inflammations, and it is of course entirely possible that the morbid process should be aborted at this stage. Indeed, it is a matter almost of certainty that in some fatal cases of tetanus the pathological action has not gone beyond the hyperæmic stage, and hence the absence of lesions in the cases examined by Billroth and others. But an hyperæmia of this kind is of course as much of the nature of inflammation as though the process had reached its full development.

How does a wound of the extremity or trunk of a nerve cause tetanus? It has been supposed by some authors that there was a neuritis in each case which advanced centripetally till it reached the spinal cord. In regard to this point, Mitchell[3] says:

"There is a prevalent belief that tetanus is more apt to arise when large nerves are slightly hurt than on other occasions; but, although there are on record many cases where this terrible malady has followed the inclusion of nerves in ligatures, in the mass of tetanic histories the causal irritation has arisen in the extreme distribution of nerves, and where there has been no proof of precedent injury to large trunks. Were it otherwise, I must more often have seen tetanus, whereas, in two hundred recorded instances of wounds of great nerves which passed under my eye during the war, not a single case of lock-jaw was seen, although in perhaps one-half, the injuries were recent, and we actually witnessed a part of the process of healing. In fact, the tendency toward irritation resulting in spasm seems to increase as the nerves divide and approach the skin. Brown-Séquard succeeded once in causing tetanus by leaving a rusty tack in the foot of an animal. I have never been able to get this result by any method, nor, in some seventy sections or wounds of nerves in animals, have I ever encountered it."

---

[1] *Op. cit.*, p. 359.

[2] "Convulsions pendant un mois chez une grenouille empoisonée par la strychnia; intégrité complète de la moëlle épinière," *Archives de Physiologie*, 1868, p. 306.

[3] "Injuries of Nerves and their Consequences," Philadelphia, 1872, p. 147.

The experience of Dr. Mitchell on this point is sufficient to determine it against the existence of a neuritis extending to the cord. Were there any such cause it would undoubtedly be more apt to arise from a wound of the trunk of a nerve and to extend to the cord, than from an injury of the terminal extremities. Moreover, the facts that tetanus has been known to follow in a few minutes after the reception of a wound, and that there is no pain along the course of the nerve, are directly at variance with the idea of a peripheral and ascending neuritis as the cause of the spinal lesions.

Treatment.—There is scarcely a sedative or stimulant remedy in the pharmacopœia which has not been employed and recommended in tetanus. Aconite, ether, belladonna, chloroform, cannabis Indica, conium, opium, tobacco, Calabar bean, ice, counter-irritants, alcohol, and many other substances, have been used, and cases reported which have apparently recovered under their administration. Then, of surgical means, excision of the injured nerve and amputation of the wounded member have also been recommended, but are not, I believe, practised now. Latterly the bromide of potassium and hydrate of chloral have been employed with favorable results.

A case in which the latter agent was successfully used in tetanus is reported by Dr. Wirth,[1] of Columbus, Ohio. In about a month the patient took nine ounces and two drachms, in doses of from thirty to forty grains, at times as often as every one and a half hour. In this case opium in large doses had been administered without effect. A number of other cases, in which chloral was administered, are cited in the same number of the *New York Medical Journal* in which Dr. Wirth's case appears, in several of which it was successful.

A very thorough analysis by my friend Dr. D. W. Yandell,[2] of Louisville, of an unpublished report on tetanus, by Dr. R. O. Cowling, embraces so much valuable information on the subject that I quote the summary entire. The term *acute* is applied to tetanus occurring within nine days of the injury, and chronic to cases ensuing after nine days:

" *Calabar bean* was given in thirty-nine cases, with thirty-nine per cent. of recoveries. Of these reported cures, but one was of acute tetanus; five others were in cases which recovered before the expiration of fourteen days. *Per contra*, there were ten deaths from chronic tetanus.

---

[1] *New York Medical Journal*, November, 1870, p. 419.

[2] *American Practitioner*, September, 1870, p. 152.

"*Indian hemp* used in twenty-five cases, with sixty-four per cent. of recoveries, of which three cases were acute, and six recovered before the symptoms lasted fourteen days.

"*Chloroform* relieved seventy per cent. of thirty-five cases, nine of which were acute, and eight recovered before fourteen days. Three chronic cases died, and two after symptoms lasted fourteen days.

"*Ether.*—Sixty per cent. of fifteen cases recovered; five acute; seven inside of fourteen days. One chronic case died.

"*Opium.*—Fifty-seven per cent. of one hundred and sixty-five cases recovered; twenty-two acute; twenty-nine before the fourteenth day. Twenty-six chronic cases were lost, and four after the disease had continued fourteen days.

"*Tobacco* relieved fifty per cent. of forty-one cases; six acute; six before fourteen days of the disease. Four chronic cases died, and one after fourteen days.

"*Quinine.*—Seventy-three per cent. of fifteen cases recovered; one acute; three before fourteen days. Three chronic cases ended fatally, and one after fourteen days' duration.

"*Aconite.*—Eight per cent. of fourteen cases recovered; none acute; none recovered before fourteen days. Death in one chronic case.

"*Stimulants.*—Eighty per cent. of thirty-three cases recovered; four acute; six within fourteen days. Six chronic cases died, and three after fourteen days.

"*Mercury.*—Fifty-seven per cent. of seventy-five cases got well; twelve before fourteen days. Seventeen chronic cases were lost, and two after fourteen days.

"*Bleeding.*—Fifty-five per cent. of fifty-eight cases recovered; nine acute; ten before the fourteenth day. Seven chronic cases were lost, and two after fourteen days.

"*Cold Affusion.*—Seventy-three per cent. of eleven cases recovered; three acute; three before fourteen days. Two chronic cases died.

"*Ice-bags.*—Seventy-seven per cent. of nine cases recovered; one acute; two in less than fourteen days.

"*Amputation.*—Sixty per cent. of seventeen cases recovered; four acute; four in less than fourteen days. Three chronic cases died, and one after fourteen days.

"*Division of nerve* relieved seventy-five per cent. of three cases; one acute; one before the fourteenth day. One chronic case died.

"*Purgatives.*—Sixty-six per cent. of seventy-four cases recovered; thirteen acute; twelve before fourteen days. Ten chronic cases died, and three after fourteen days.

"*Turpentine* relieved seventy per cent. of sixteen cases; six acute; four before fourteen days. Five chronic cases died, and two after fourteen days."

Among the conclusions arrived at by Dr. Yandell from these data are, that "recoveries from traumatic tetanus have been usually in cases in which the disease occurs subsequent to nine days after the injury; that when the symptoms last fourteen days recovery is the rule, and death the exception, apparently independent of the treatment; that chloroform, up to this time, has yielded the largest percentage of cures in acute tetanus; that the true test of a remedy for tetanus is its influence on the history of the disease: does it cure cases in which the disease has set in previous to the ninth day? does it fail in cases whose duration exceeds fourteen days? and that no agent, tried by these tests, has yet established its claims as a true remedy for tetanus."

It is, perhaps, scarcely necessary to say that I fully accord with these opinions.

Judging from its effects upon the spinal cord, it was supposed by Mr. Morgan that woorara injected into the blood might prove efficacious in tetanus. Experience, however, has not confirmed this view; and the researches of Dr. Cowling show that it is one of the most inefficient of remedies.

In a case which was under my charge fifteen years ago, when I was one of the surgeons of the Baltimore Infirmary, I injected corroval—a remedy which the investigations of Dr. Mitchell and myself had proved to be antagonistic to strychnia—into the blood. The patient, a colored boy, became affected with tetanus two days after his arm had been amputated by my friend and colleague Prof. Nathan R. Smith. Cannabis Indica, morphia, and chloroform, had been used without effect, when at my request Prof. Smith turned the case over to me, in order that corroval might be administered. Two drops of a strong solution of the substance in water were injected into the cellular tissue of the forearm. At the time the pulse was 160, and the respirations about 75. There was very decided opisthotonos. In three minutes the pulse had fallen to 152. Two more drops were then injected, and the pulse fell to 144. As it soon rose again, two more drops were injected, when it fell to 132, and the respirations to 64. The spasms still continuing, two more drops were injected. In five minutes the pulse began to decline rapidly, and in ten minutes had fallen to 90. At this time the patient had a violent tetanic spasm, and during its continuance the pulse became intermittent. It then rapidly went down to 40, then to 30, and during a violent spasm the patient died. From this record it will be seen that at no time did the corroval exercise the least effect over the disease.[1]

---

[1] "Traumatic Tetanus; Inoculation with Corroval; Death," by Edward Milholland, M. D., Resident Physician at the Baltimore Infirmary. In *Maryland and Virginia Medical Journal*, January, 1861, p. 13.

As I have stated, three successful cases have occurred in my practice. One of these I saw in consultation with Dr. J. Lewis Smith, of this city. It was traumatic, and had ensued two weeks after a wound of the foot by a nail. The patient was treated by cannabis Indica, and the persistent application of ice to the spine. The spasms were greatly lessened in force and frequency, and recovery took place within two weeks. Another, which was also traumatic and acute—that is, making its appearance within nine days after the injury—was treated according to the same plan, and recovered in sixteen days, though the jaws remained stiff for several weeks afterward. The wound was caused by an ice-pick being accidentally thrust through the hand. The third case was that of an eminent musician of this city, who, while drilling with the regiment to which he belonged, injured his thumb with a splinter from the stock of his rifle. The first evidence of tetanus appeared on the twelfth day. The attack was not very severe. I administered the extract of cannabis Indica (Squires's) in doses of half a grain every two hours, and kept up the application of ice to the spine continuously for six days. There were several violent spasms during this period, and the opisthotonos was well marked. At the end of a week the cannabis Indica was omitted for a day, but, the spasms becoming more frequent and severe, it was resumed as before, and continued with tolerable regularity for ten days longer. During this period there were but two spasms, and the opisthotonos became less. It was then gradually diminished, and on the twenty-fifth day was left off altogether, the patient being convalescent.

I am disposed to think that, whatever internal medication be adopted, the application of ice to the spine is a measure which should always form a feature of the treatment.

IV.

INFLAMMATION OF THE ANTERIOR COLUMNS OF THE SPINAL CORD (SCLEROSIS OF THE COLUMNS OF TÜRCK).

Türck[1] has shown that the anterior columns of the spinal cord are subject to a chronic inflammation such as is now known under the name of sclerosis. In the cases which he described the morbid process involved symmetrically a small region on each side of the anterior median fissure—that part which is designated the column of Türck. Microscopical examination showed proliferation of the neuroglia, with degeneration of the true nerve-elements.

---

[1] "Ueber Degeneration einzelner Rückenmarksstränge, welche sich ohne primäre Krankheit des Gehirnes oder Rückenmarks entwickelt," "Sitzungsberichte der Kaiserlichen Academie der Wissenschaften," Mat. nat. Cl., 1856, p. 112.

The course of the disease, the symptoms, causes, etc., together with the morbid anatomy and pathology, do not differ essentially from the corresponding affection of the lateral columns which is more frequent and is more thoroughly understood. Our present knowledge leads us to the inference that the columns of Türck are in function similar to the lateral columns. The number of cases in which they have been found altered is as yet small, and they have not been very thoroughly worked up. In some cases they have been sclerosed in conjunction with a like condition of the lateral columns.

I shall, therefore, pass at once to the consideration of the next division of the subject.

V.

INFLAMMATION OF THE LATERAL COLUMNS OF THE SPINAL CORD; SPASTIC SPINAL PARALYSIS (PRIMARY SYMMETRICAL LATERAL SCLEROSIS).

Türck,[1] who, as we have seen, demonstrated the fact that the anterior columns of the cord could be the primary seat of sclerosis without any other region participating in the lesion, also showed that the lateral columns could be similarly affected. Türck's investigations were allowed to remain scarcely noticed for ten years, when Charcot[2] made like observations and since then has aided in establishing it as a distinct pathological condition.

Symptoms.—The chief phenomena of the disease under consideration are paralysis and contraction of the affected limbs. The lower extremities are, more than the upper, liable to be the seat of these symptoms. The loss of power is very gradual, and there is no atrophy beyond the general emaciation consequent upon diminished use of the muscles. Sensibility is not in general affected, but in some cases there is more or less pain in the paretic limbs and in the back near the seat of the disease.

The paralysis is rarely complete. At first the patient merely tires more readily, slight exertion fatigues him, and this is especially noticed in the muscles which flex the leg upon the thighs, and the consequent sensation of weariness is experienced in the popliteal space. Sometimes it is shown in the sudden relaxation of the extensor muscles of the leg and the fall of the patient thereby; at others, in the fact that the extensors of the foot become weak, allowing the toes to drop, and hence causing stumbling. The gait then becomes characteristic. Owing to the fact that the patient's extensor muscles are weak, he is unable to lift the feet high enough to cause them to clear the ground, and hence he throws them out by means of the abductor muscles of the thigh, and thus causes them to describe an arc of a circle. Then in putting them down the heel strikes the ground a longer time before the sole than i does in the natural gait, and hence the foot comes down with a jerkin motion. This is the ordinary manner of walking practised by a person affected with the disease under notice. In another form of locomotion, the body is moved laterally on the thighs, first to one side and then to the other, in such a way as to cause the feet to be raised high enough without the complete action of the extensor muscles. The gait is therefore similar to that of a duck, or of a woman with a very wide pelvis. The motion of the body is almost serpentine, and the feet glide over the ground barely lifted high enough to avoid contact.

---

[1] *Op. cit.*, p. 112.

[2] *L'Union Médicale*, 1855.

In both the methods of walking the patient requires support. At first a cane answers, then he comes to crutches, and eventually the assistance of an attendant becomes necessary.

As a consequence of the paralysis, and the contractions which eventually ensue, the movements are often complicated and sometimes rendered impossible by the legs becoming interlocked at every attempt to walk. In a patient from Connecticut under my care, not long since, this difficulty was a very prominent feature, and though the muscles of flexion and extension were sufficiently strong to allow of his walking, those which abduct the thighs were so materially paralyzed, and the adductors were so greatly contracted, as to produce the condition mentioned.

Reflex movements, so far from being lessened, are generally exalted; and this is especially true of the "tendon reflex," as exhibited when one leg is crossed over the other and a smart blow given with the edge of the hand over the tendon of the quadriceps extensor, just above the patella. The leg is suddenly extended, and bounds much higher than it normally does under like excitation. The electric contractility of the muscles remains unimpaired.

Sometimes the contractions, which are so prominent a feature, relax, but they again supervene, and generally persist with more or less intensity till the closing stages of the disease, when the power of the cord becoming exhausted entirely, and all the muscles being paralyzed, the spasmodic action ceases.

A very remarkable case is one reported by Charcot,[1] of a woman who, after several hysterical attacks, was seized, after having been greatly frightened, with a violent paroxysm of hysteria, which was soon followed by a general trembling, accompanied by a weakness of the limbs. At the end of a month the feebleness was such that she could not leave her bed. About the same time the trembling ceased, but was succeeded by a contraction which affected at first the extremities of the left side, but in the course of three weeks involved those of the right side also. The neck also became rigid.

---

[1] Cited by Bourneville, "De la contraction hystérique permanente," Paris, 1872, p. 77.

All these phenomena persisted, they even increased so that in the early part of 1850 she was admitted into the Charité.

At that time she was confined to her bed in the dorsal decubitus, not being able to move her limbs. Her general health was good, and her cerebral functions were normal. The muscles of the neck were painful and stiff. The skin on the left anterior part of the thorax was hyperæsthetic, the condition being exactly bounded by the median line. The tactile sensibility was a little obtuse in the left superior extremity, but the sensibility to pain was exaggerated. The muscular sensibility was also more marked than in the normal state.

The superior extremities were strongly contracted; the forearm was flexed on the arm, and the fingers were also strongly flexed. Attempts made to extend the limbs were only partially successful and caused pain. The contracted muscles were the seat of continuous spontaneous pains, and from time to time sudden movements took place in these members, either spontaneously or as a consequence of reflex action. Neither of these limbs could be moved by voluntary power.

The trunk was rigid and its muscles were painful to pressure. Pressure on the cervical region of the skin also caused pain.

Both inferior extremities were also strongly flexed. Pressure on the muscles caused pain, and there were also darting pains through these limbs.

The case was regarded as one of hysteria. The patient remained in the hospital two years, and left in about the same state as when she entered. Subsequently the symptoms almost entirely disappeared, nothing remaining but a weakness of the lower extremities and a slight degree of contraction of the upper. But in 1855 she had another hysterical attack, and this was followed by a return of the former condition.

In 1856 she entered the Salpêtrière, and in 1862 her case was studied by M. Charcot. The symptoms were similar to those which have been described, though even more pronounced. In 1864 she died during an attack of erysipelas.

The post-mortem examination showed the essential lesion to consist of sclerosis of the lateral column from the medulla oblongata to the lower boundary of the lumbar enlargement. The gray matter was healthy throughout.

A study of this case shows that the principal symptoms of primary symmetrical lateral sclerosis are as Türck described them in his memoir —paralysis, contractions, and pain in the back and limbs.

Causes.—The causes of the disease are probably similar to those producing so many other spinal affections—cold, dampness, over-exertion, syphilis. Nothing very definite is known on the subject.

Diagnosis.—The elements of the diagnosis of primary symmetrical lateral sclerosis of the spinal cord are the presence of contractions with paralysis but without atrophy, and the absence of any organic disease of the brain or superior part of the cord (bulb) which could give rise to the condition as a secondary disorder. It must, however, be borne in mind that contractions are the expression of degeneration of the lateral columns. It is only by attention to the clinical history of the case that we can ascertain whether the lesion is primary or secondary.

The distinction between the disease in question and progressive muscular atrophy is so clear as scarcely to require comment, and from amyotrophic lateral spinal sclerosis, the absence of atrophy and its accompaniments, and of a tendency to attack the nuclei of the bulbar nerves, will serve to make the discrimination.

It has more affinities, so far as its symptoms are concerned, with chronic spinal meningitis, multiple spinal sclerosis, and with tumors which, by their pressure on the cord, may give rise to very similar phenomena to those exhibited in lateral sclerosis. I am afraid the difficulties of making a diagnosis between it and these affections are, in the present state of our knowledge, almost insurmountable. I know of no sure signs by which the discrimination can be made.

Prognosis.—Although remissions may take place, the prospect of an entire cure is not very great. The progress of the disease is, however, slow in the majority of cases, and its course may, I am satisfied, be materially retarded if not altogether arrested in some cases.

Morbid Anatomy and Pathology.—Türck was the first to associate sclerosis of the lateral columns with a definite set of symptoms. In three of the cases of the twelve on which his memoir is based he found these regions of the cord the seat of symmetrical sclerosis. It has been very definitely settled that the lateral columns are, in embryonic life, anatomically distinct from the rest of the cord; and, though in the process of development this anatomical separation is apparently lost, pathology shows us that it in reality exists.

Charcot,[1] in considering this subject, calls attention to the fact that transverse sections of the cord in cases of primary symmetrical lateral sclerosis made through the cervical enlargement show that the alteration embraces a greater extent of the cord than when any other part is affected. Thus, when the region in question is the seat of the lesion the sclerosis extends anteriorly as far as, and even beyond, the external angle of the anterior horn, while posteriorly it almost encroaches on the posterior tract of gray matter. On the outside it is always separated from the cortical layer by a tract of white tissue which remains intact.

In Fig. 73 is represented a transverse section of the cord made through the cervical enlargement. *a* denotes the sclerosed portion extending beyond the external angle of the anterior tract of gray matter reaching to the posterior tract behind, and separated from the cortex by a layer of unaltered white substance.

---

[1] *Op cit.*, p. 220.

FIG. 73.

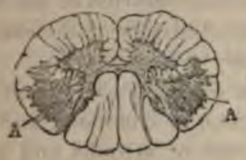


FIG. 74.

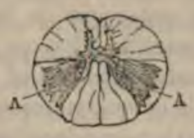
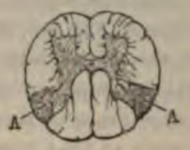


FIG. 75.

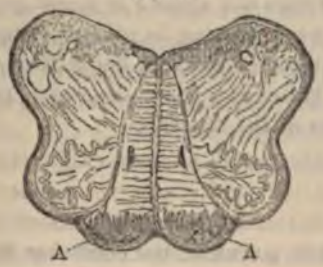


In the dorsal region the lesion is more circumscribed, as is readily seen from an examination of Fig. 74, which represents a section of the cord through the middle of that part. In front it scarcely reaches the posterior boundary of the anterior tract of gray matter. As in the section just described, the sclerosed portion does not extend to the cortical layer of the cord.

In the lumbar region the lesion is still less extensive, occupying only about a quarter of the area of the lateral columns. Unlike the lesion in the cervical and dorsal regions, it touches the cortical layer of the cord (Fig. 75).

Finally, in those cases in which the morbid process extends to the medulla oblongata, we find it seated in the anterior pyramids, not in the nuclei of the bulbar nerves, as in cases of amyotrophic lateral spinal sclerosis to be presently considered. Fig. 76 represents a transverse section of the medulla oblongata through the middle part of the olivary bodies; *A A*, the anterior pyramids in a state of sclerosis.

FIG 76.



In primary symmetrical lateral sclerosis, the initial stage, as in other inflammatory affections of the spinal cord, whether acute or chronic, is probably congestion.

Treatment.—In the early period of the disease large doses of ergot will rarely fail to be of service. I have several times succeeded in relieving the paralysis and arresting the spasms of the limbs in cases presenting all the initial phenomena of lateral sclerosis, by the persistent and free use of this remedy. But to be efficacious it must be given in the very first stage, before the paralysis becomes extreme, or permanent contractions are present. A drachm of the fluid-extract three times a day is the smallest dose likely to prove efficacious.

If there is reason to suspect the influence of syphilis in producing the disease, the iodide of potassium in large and gradually-increasing doses should be administered. Charcot and Gombault[1] have proved the existence of disseminated sclerosis of the cord in a woman affected with syphilis, and there is of course reason to believe that the diffused form such as that now under consideration may have a like origin.

In such cases mercury may also be given, preferably in the form of the bichloride, with the iodide of potassium.

Later, no treatment is, so far as we know, calculated to materially arrest the progress of the disease. I have employed the chloride of barium, with apparently some temporary advantage. Nitrate of silver and cod-liver oil have also occasionally improved the strength of the patient and lessened the rigidity of the contractions, but only for a short time; and the primary uninterrupted galvanic current to the spinal column and the contracted muscles has also proved serviceable in the same way and to a like extent.

Up to quite a recent period I had never derived any benefit in cases of lateral sclerosis from counter-irritation, but I am disposed to think from some late experience that the actual cautery, applied on each side of the spinous processes throughout the entire length of the vertebral column and frequently repeated, is useful.

For the relief of pain, morphia may be administered, or what is I think preferable, as it does not appear to be an excitant of the cord, codeine. Half a grain or more may be given as required.

Hypodermic injections of atropia, beginning with the one hundred and twentieth of a grain and increasing gradually, are beneficial in mitigating the spasms of the muscles.

VI.

INFLAMMATION OF THE LATERAL COLUMNS OF THE SPINAL CORD AND OF THE ANTERIOR TRACT OF GRAY MATTER (AMYOTROPHIC LATERAL SPINAL SCLEROSIS).

For the recognition of this affection and the patho-anatomical data relative to its identity, we are indebted to Charcot, who, with his customary ability, has presented a mass of facts abundantly sustaining his views in regard to its autonomy. Cases exhibiting the phenomena of amyotrophic lateral spinal sclerosis were noticed, and their details published before he enunciated his doctrines on the subject; but the relations of the lesions to the symptoms were not known previous to his observations.

---

[1] "Note sur une cas des lésions disséminées des centres nerveux observées chez une femme syphilitique," *Archives de physiologie*, 1873, p. 148.

Symptoms.—The first symptom to make its appearance in the affection under notice is paralysis, which occurs ordinarily gradually, advances steadily, and may involve at the same time one or more of the limbs. Generally atrophy ensues soon after the appearance of the paralysis, and, as in infantile spinal paralysis, and the spinal paralysis of adults, involves whole groups of muscles at once—not the individual muscles in succession, as in progressive muscular atrophy.

After a time the morbid process in its ascending course reaches the medulla oblongata, and, thus implicating the nuclei of the facial, spinal accessory hypoglossal, and pneumogastric nerves, especially the two latter, causes atrophy of the tongue, and many of the other symptoms met with in progressive muscular atrophy affecting these centres. Finally, death takes place from interruption to the processes of respiration and circulation.

The muscles which are the seat of atrophy are subject, as in progressive muscular atrophy, to fibrillary contractions, which, however, as in the last-named affection, precede the atrophy, and advance and attain their greatest development *pari passu* with the wasting.

The electro-muscular contractility remains unaffected in these muscles, which are the seat of the atrophy so long as any muscle remains to contract.

But the feature which is most characteristic of amyotrophic lateral spinal sclerosis is the permanent contractions of which the affected limbs are the seat. These, though in part due, as Charcot[1] says, to the paralysis of certain antagonistic muscles, are mainly caused by spasmodic contractions of the non-paralyzed or partially paralyzed muscles, so that the joints are rigidly flexed. The position assumed when the forearm and hand are the seat of this deformation, is shown in Fig. 77. The fingers are flexed upon the palm, the thumb adducted, and the hand strongly bent upon the arm.

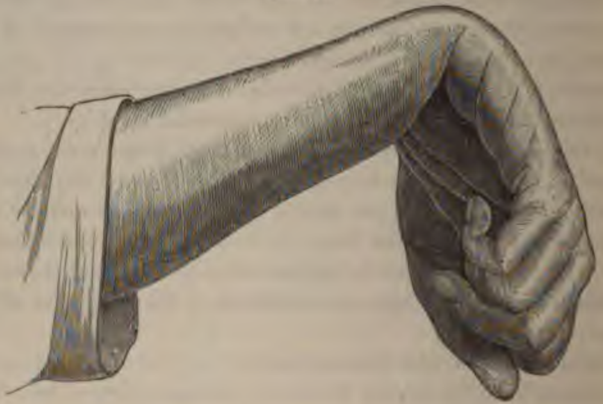
In the case of a gentleman who came under my observation in September, 1874, the position of the left hand was very similar to that shown in the figure. The arm was semiflexed, and the whole member held firmly against the walls of the chest, by the action of the pectoral and latissimus dorsi muscles. Any attempt to overcome the contractions was strongly resisted by the muscles, and caused very considerable pain. The atrophy of the paralyzed muscles was well marked, and fibrillary contractions were easily excited, even if not present when the inspection was made.

In this case the disease had appeared suddenly six months previously, after exposure subsequent to a debauch. The upper extremities only were affected, but there was, even when I saw the patient, a little restraint in the movements of the tongue. I did not see him again, but I heard that death had ensued from dysentery three months after

---

[1] "Leçons sur les maladies du système nerveux," 3me. fascicule, Paris, 1874, p. 234.

FIG. 77.



his visit to me. I also learned that his tongue had become atrophied, and that there was difficulty of swallowing.

In another case the contracted muscles were the pectorals, and the left arm was, in consequence, drawn strongly across the front of the chest. This patient, a man forty years of age, was paralyzed in both upper extremities, but the contraction, when I first saw him, was limited, as stated, to the pectoral muscles. He visited me again about six months afterward, and then the right upper extremity was also contracted throughout its extent. The fingers were bent on the palm to such a degree that they could not be opened by any force which it was safe to apply, and pressed so strongly on the palm as to cause pain. If the nails were allowed to project beyond the ends of the fingers—and it was very difficult to keep them short—they entered into the skin and caused painful sores. The hand was flexed on the wrist, the elbow half bent, and the arm was held firmly against the side of the chest. At the time of the first visit of this patient there was no evidence of any alteration of the medulla oblongata, but at the second visit there were several indications of incipient bulbar paralysis. Deglutition was effected with difficulty, the tongue could not be carried to the roof of the mouth, was protruded only slightly upon great effort being made, and was the seat of constant fibrillary contractions.

The atrophy of the paralyzed muscles was well marked, and fibrillations were so strong as to be a source of great discomfort. The lower extremities were not then involved, and the bladder and sphincters were intact. I have not seen the patient since—now about seven months—and am ignorant of the subsequent course of the disease.

The contractions are not always similar, either in extent or strength, in the corresponding limbs, and they may for a time, especially in the early stages of the disease, disappear. But they reappear later, and tend to become more and more rigid as the affection advances; still, in the most extreme period of the malady, as the atrophy becomes pronounced, they disappear wholly or in part, there being little if any muscle left to maintain the contraction.

Another feature is a spasmodic extension of the paralyzed limbs, especially the lower, which is most strongly marked as the patient lies in bed; or the members may be involuntarily flexed and remain in that condition for several minutes or longer. These movements are not ordinarily accompanied with pain, as are those of spinal meningitis, which in many respects they resemble.

The patient very generally experiences severe pains in the affected limbs, which are aggravated or excited by pressure or traction in the muscles. Numbness is also usually present to a greater or less degree, but there is never complete anæsthesia.

A peculiar kind of tremor is sometimes seen in the limbs, the muscles of which are partially paralyzed and atrophied. This is in reality not so much a tremor as it is a more extensive movement, resembling that which is present in some old cases of hemiplegia from cerebral hæmorrhage. It is only, like that, manifested when voluntary movements are attempted.

As previously stated, the disease, unless death ensues from some intercurrent affection, eventually extends to the medulla oblongata. In none of my cases has this circumstance been present to any marked degree while the patients were under my observation; but in two, as we have seen, there were indications of such extension when they passed from under my notice. Charcot states it to be an invariable sequence, so far as his observations extend, and he sums up the subject as follows: The paralysis of the tongue, inducing a difficulty of swallowing and of articulation, may cause a complete loss of the power of speech. The paralyzed tongue presents very soon, in general, a certain degree of atrophy; it is shrunken, wrinkled, and agitated with vermicular movements.

The paralysis of the veil of the palate makes the voice nasal, and, with the laryngeal paralysis, renders the act of deglutition difficult.

The orbicularis oris being paralyzed, an alteration in the form of the countenance takes place. The mouth is considerably enlarged transversely, through the predominance of the action of the muscles which are not involved. The naso-labial furrows are depressed. The symptoms give to the physiognomy a sad expression. The mouth, especially after laughing or weeping, remains for a long time half open, and allows the viscid saliva to flow continually.

Finally, by reason of the implication of the pneumogastrics, grave troubles of the respiration and circulation supervene, and cause the death of the patient, already weakened by insufficient nourishment.

In some cases there are variations from the ordinary course of the disease. It has begun in the lower extremities instead of the upper, and again has been restricted in its domain for a long time to a single limb or to one side of the body. In two cases, according to Charcot, it has begun with the bulbar symptoms, which in general appear only at the end. In regard to such instances as these last, there is doubt of their being examples of amyotrophic lateral sclerosis.

Causes.—Exposure to cold and dampness appears to be the most efficient exciting cause of the disease. In one of Charcot's[1] cases, the patient, an itinerant showman, was exposed during a journey to cold and rain. The following morning he was taken with a chill, which was repeated thirty-six hours afterward, and then he was seized with pains along the course of the nerves, and in the joints mainly of the upper extremities. Fibrillations accompanied them, and paralysis and atrophy soon followed. In the case occurring in my own experience, the patient became intoxicated, and wandering into the Central Park, lay all night on wet grass, exposed to a cold, drizzling rain. In the morning he was arrested and taken to the police court, and sent to Blackwell's Island for ten days. On his way up, he was subjected to the influence of a cold wind, which, blowing on his wet clothes, chilled him more and more. The following morning he was discharged, his friends having ascertained his situation and paid his fine. But he already felt a degree of weakness in his arms, and in the course of a week they were to a great extent deprived of motor power. Fibrillary contractions were present from the first, but there was no pain anywhere.

In the majority of cases no cause can reasonably be assigned. There appears to be no hereditary influence to the disease.

Diagnosis.—The diagnosis of amyotrophic lateral spinal sclerosis presents many features of interest. A consideration of the essential phenomena shows that they are as follows :

1. Paralysis occurring in symmetrical parts of the body, unaccompanied by anæsthesia.
2. Atrophy following the paralysis, and attacking masses of muscles.
3. Spasmodic rigidity, eventually leading to permanent contractions, lasting up to the last stage of the disease.
4. Extension of the affection to the lower extremities, and the supervention of intermittent and tonic contractions or rigidity.
5. The implication of the medulla oblongata, and death in the course of two or three years.

Thus, we see that the affinities of amyotrophic lateral spinal sclerosis are with spinal paralysis of adults and progressive muscular atrophy with which latter disease it was confounded by Dumesnil,[1] Charcot,[2] and others, up to quite a late period.

---

[1] "Deux cas d'atrophie musculaire progressive," *Archives de physiologie*, 1869, p. 637

But the differences between it and both these diseases are sufficiently striking to prevent much danger of confounding them. Thus from spinal paralysis of adults it is discriminated by the facts that reflex excitability is not impaired, nor the electric excitability of the muscle diminished, as in the former affection; that the atrophy is more profound and constant; that fibrillations are present, and especially by the existence of the spasmodic contractions of the limbs which form so prominent a feature of amyotrophic lateral spinal sclerosis.

From progressive muscular atrophy the distinction can be readily made out. In the facts that paralysis precedes the atrophy, that the wasting takes place in groups of muscles, and that spasmodic contractions occur in amyotrophic lateral spinal sclerosis; while in progressive muscular atrophy the paralysis is consequent on the wasting, the muscles shrink singly and irregularly, fibre by fibre, and spasmodic contractions do not occur, we have sufficiently precise diagnostic marks of differences between the two affections.

Prognosis.—There is no case of cure on record. The course of the disease is progressively onward. In the majority of cases the fatal termination occurs within two years; occasionally, it is deferred for a few months longer.

Morbid Anatomy and Pathology.—As I have said, amyotrophic lateral spinal sclerosis has, until quite lately, been regarded as, at most, an eccentric form of progressive muscular atrophy. It is among the reports of cases of this affection, therefore, that we must search for early data relative to the morbid anatomy of the disease under consideration.

Dumesnil,[3] in 1867, reported the details of five cases of spinal disease under the name of progressive muscular atrophy, two of which were, undoubtedly, instances of amyotrophic lateral spinal sclerosis. In both of these, symptoms such as have been described were present, and, on post-mortem examination, lesions of the lateral columns and anterior horns of gray matter were found to exist.

Charcot and Joffroy[4] have given with fullness of detail the particulars of two cases of amyotrophic lateral spinal paralysis, in which the post-mortem examination was very thorough.

---

[1] "Nouveaux faits relatifs à la pathogenie de l'atrophie musculaire progressive," *Gazette hebdomadaire*, Nos. 27, 29, 30, 1867.

[2] "Deux cas d'atrophie musculaire progressive avec lésions de la substance grise et des faiseaux antéro-latéraux de la moëlle épinière," *Archives de physiologie*, No. 4, 1869.

[3] "Nouveaux faits relatifs à la pathogenie de l'atrophie musculaire progressive," *Gazette hebdomadaire*, Nos. 27, 29, 30, 1867.

[4] "Deux cas d'atrophie musculaire progressive avec lésions de la substance grise et des faiseaux antéro-latéraux de la moëlle épinière," *Archives de physiologie*, 1867.

The first of these is reported as a case of progressive muscular atrophy, especially marked in the upper extremities, with atrophy of the muscles of the tongue and of the orbicularis oris and paralysis with rigidity of the inferior limbs. On post-mortem examination the nerve-cells of the anterior horns in the cervical and dorsal regions were found atrophied, while many had disappeared. In the bulb there were atrophy and disappearance of the nerve-cells, constituting the nucleus of the hypoglossal. The anterior roots of the spinal nerves and the roots of the hypoglossal and the facial were also atrophied. In addition, there was symmetrical diffused sclerosis of the lateral columns. In this case the bulb was affected after the lower parts of the cord, and the lesion of the nuclei of the hypoglossal and facial was of such a nature as to cause atrophy of the tongue and orbicularis-oris muscle. The instance was, therefore, of a typical character.

The second case has already been cited under another head of this chapter. It is entitled—Progressive muscular atrophy especially manifested in the upper extremities; acute pains in the limbs coming on in paroxysms; anæsthesia in certain parts of the body; paralysis with rigidity of the inferior extremities; lesions of the nerve-cells of the anterior horns of gray matter; centres of gray degeneration in the posterior horns; symmetrical diffused sclerosis of the lateral columns; considerable thickening of the spinal dura mater and pia mater of the cervical enlargement.

This case was not an uncomplicated one, but still the essential lesions of the anterior horns of gray matter and of the lateral columns are perceived to have been present.

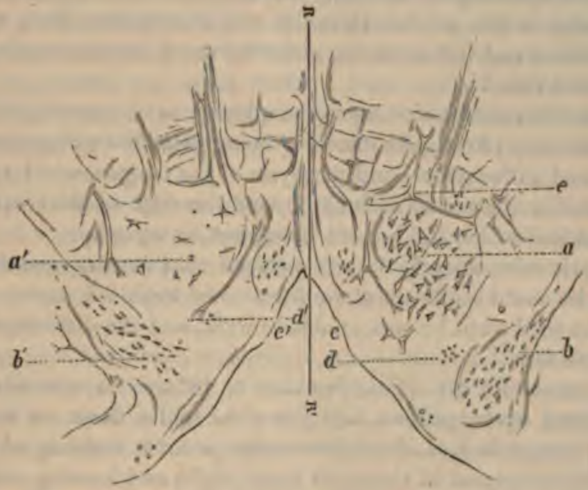
Gombault[1] reports the case of a woman in whom the symptoms were not developed with any suddenness, but in whom there gradually supervened loss of power with atrophy and contractions in the upper extremities, then paralysis with atrophy in the lower limbs, and finally atrophy of the muscles of the tongue and lips, with difficulty of swallowing, and the other symptoms of glosso-labio-laryngeal paralysis. Post-mortem examination showed the lateral columns to be symmetrically sclerosed, and in the anterior horns of gray matter symmetrical lesions, exactly limited to this region, and consisting of atrophy, pigmentary degeneration, and disappearance of the nerve-cells. In the bulb, the nuclei of origin of the bulbar nerves were similarly altered.

It is perceived, therefore, that in amyotrophic lateral spinal sclerosis the essential lesions are seated symmetrically in the lateral columns and in the anterior horns of gray matter, and that when the morbid process extends—as it always does, if the patient does not die in the mean time of some intercurrent affection—to the medulla oblongata, it invades the nuclei of origin of the nerves which are affected in glosso-labio-laryngeal paralysis. The accompanying woodcut from Charcot represents a section of the bulb made at the level of the middle part of the nucleus of the hypoglossal; *a* *b* to the right of the imaginary line R R′ represents the normal condition; *a*, the nucleus of the hypoglossal composed of about thirty multipolar ganglion-cells; *e*, a vessel circumscribing the nucleus; *c*, the floor of the fourth ventricle; *d*, the *fasciculus teres*; *b*, nucleus of the pneumogastric. On the left the letters *a′* *b′*, etc., represent the corresponding parts in a case of amyotrophic lateral spinal sclerosis. It is perceived that only five or six cells exist in the nucleus of the hypoglossal. The nucleus of the pneumogastric is, on the contrary, normal.

---

[1] "Sclérose symétrique des cordons latéraux de la moëlle et des pyramides antérieurs dans la bulbe; atrophie des cellules des cornes antérieures de la moëlle; atrophie musculaire progressive; paralysie glosso-laryngée," *Archives de physiologie*, 1872, p. 589.

FIG. 78.



Now, it appears to me that M. Charcot is wrong in considering, as he apparently does, every case of glosso-labio-laryngeal paralysis accompanied by progressive muscular atrophy of the muscles lower down, as one of primary amyotrophic lateral spinal sclerosis.

Those cases of glosso-labio-laryngeal paralysis which, at a later period, exhibit the phenomena of progressive muscular atrophy in the muscles of the upper extremities and of other parts of the body are, in my opinion, not cases in which there is primitive lesion of the lateral columns, but examples of secondary degeneration of the cord, being produced as a consequence of the superior lesion.

The case reported by Dr. Hun,[1] of Albany, is quoted by Charcot[2] as an instance of amyotrophic lateral spinal sclerosis, but, according to the view above expressed, it was in reality a case of glosso-labio-laryngeal paralysis with secondary degeneration of the spinal cord. The patient, a man aged fifty-eight, first noticed that the saliva dribbled from his mouth when speaking or writing. Shortly afterward he was conscious of a difficulty in the pronunciation of words, and then there were evident hesitation and defect in the articulation of certain words, and his voice became nasal.

---

[1] "Labio-Glosso-Laryngeal Paralysis," *American Journal of Insanity*, 1871, p. 194.

[2] "Leçons sur les maladies du système nerveux," Paris, 1874, p. 229

A year afterward there were complete loss of speech, difficulty of deglutition—any effort at swallowing being attended with paroxysms of coughing and suffocation—and paralysis of the tongue, which could only be protruded a quarter of an inch beyond the edge of the teeth. There was partial loss of motion in both arms, but no atrophy.

A month subsequently it was noticed that he dragged his feet a little, but he could still walk alone for a considerable distance. The paralysis advanced until he was unable to walk, and the difficulty of deglutition increased.

When seen by Dr. Hun, January 4, 1871, "he was sitting in a chair propped up by pillows, being unable to lie down on account of dyspnœa; complete loss of motion except a little nodding of the head and a little movement of the right hand; sight and hearing unimpaired; speech entirely lost; mouth partly open; and lips immovable, except a slight twitching of the left angle of the mouth; cheeks flaccid; tongue completely paralyzed and lying on the floor of the mouth; respiration feeble, and occasional coughing; pulse 90 per minute and regular; both arms paralyzed and slightly flexed, and attempts to straighten the fingers caused pain; lower extremities completely paralyzed, and feet and ankles œdematous; defecation natural; micturition slow and frequent; attempts to swallow occasioned distressing cough and suffocation, and the aliments were often rejected through the nose.

"He remained in this condition until the afternoon of the same day, when an attempt to swallow some porridge brought on severe coughing and strangling. At seven o'clock that evening he died without a struggle.

"*Autopsy* twenty hours after death.

"*External Appearances.*—Rigor mortis well marked. Body spare but not emaciated, no very evident signs of muscular atrophy.

"*Head.*—Scalp very dry. Skull-cap removed with great difficulty, owing to adhesions of the dura mater, which was torn in trying to separate it from the bone. Dura mater very much thickened. Arachnoid normal with considerable serous infiltration of the sub-arachnoidean connective tissue. Pia mater much injected. The cerebral substance, both cortical and medullary, appeared to be of normal color and consistency but exceedingly hyperæmic. The following conditions of the cranial nerves were found: 1. Olfactory normal; 2. Optic normal; 3. Motor oculi normal; 4. Patheticus small; 5. Trigeminus, on the left side flattened, gray, and softened; on the right side larger and very hyperæmic; 6. Abducens atrophied especially on the left side; 7. Facial atrophied and gray on both sides ; 8. Auditory normal ; 9. Glosso-pharyngeal normal; 10. Pneumogastric atrophied on both sides; 11. Spinal accessory much atrophied ; 12. Hypoglossal so much atrophied on both sides as to resemble mere threads or filaments of connective tissue. The corpora striata and optic thalami were normal. The cerebellum was very hyperæmic, but otherwise presented nothing unusual. The pons Varolii and medulla oblongata appeared to be of firmer consistency than usual.

"*Spinal Cord.*—Spinal meninges much injected. The anterior spinal roots were atrophied, especially on the left side. Transverse sections of the cord showed the anterior cornua of gray matter, as well as the left anterior and right lateral column, to be of a dark rose-color, as if very hyperæmic.

"Portions of the brain, cerebellum, and spinal cord were immersed in absolute alcohol, preparatory to making sections for microscopic examination. When sufficiently hardened, thin sections were made, stained with carmine, rendered transparent with benzole, and mounted in balsam.

"The sections of the brain revealed nothing abnormal. The sections of the cerebellum showed a very hyperæmic condition of the part, and a granular degeneration of the large ganglionic cells forming the middle layer of the cortical portion.

"Thirty sections were made at various levels of the medulla oblongata, involving the roots and nuclei of implantation of the cranial nerves, especially those of the facial and hypoglossal. Careful microscopic examination of these specimens, with objectives varying from fifteen to nine hundred diameters, demonstrated that the portion of medulla forming the floor of the fourth ventricle was the seat of several pathological lesions.

"There was a decided hypertrophy or overgrowth of the connective tissue, which appeared to have encroached upon and to some extent replaced the several groups of ganglionic cells which form the nuclei of implantation for the facial and hypoglossal nerves. The individual cells comprising these groups were separated from one another, and in many instances had lost their stellate appearance ; their radiating processes having been destroyed, so that each cell remained isolated and disconnected from its neighbors. These cells had also undergone a degenerative process, which in many cases rendered them simply a collection of fine granules, and a deposit of brownish-yellow pigment had taken place to such an extent as to give the cells an appearance almost precisely similar to those which are normally found in the locus niger of Soemmering ; they were fewer in number than usual and diminutive in size.

"Sections of the cord made in the cervical, dorsal, and lumbar regions, showed a sclerosis with increase of the connective tissue in the anterior and lateral columns, which was most marked in the left anterior and lateral columns. The multipolar ganglion cells, situated in the anterior cornua of gray matter, were fewer in number than usual, and many of them appeared granular and very much pigmented."

As Dr. Hun subsequently remarks, there was here "a descending degeneration of the motor tracts of the cord consecutive to a primary lesion situated in the medulla. This is fully in accordance with the views presented by Bouchard in his work on secondary degenerations of the spinal cord, and accounts for the progressive paralysis of the trunk and extremities which follows the original loss of motion in the lips, tongue, and palate."

The cases cited by M. Charcot from Leyden[1] are similar in general characteristics.

To repeat, glosso-labio-laryngeal paralysis is a paralysis without atrophy. Paralysis and atrophy consequent to it of other parts lower down, are due to secondary degenerations of the cord. Amyotrophic lateral spinal sclerosis is a paralysis with atrophy. It has a tendency to ascend and to involve the nuclei of the bulbar nerves, causing the atrophy of the muscles of the lips, tongue, and palate, and accompanied with fibrillary contractions, which latter are not phenomena of glosso-labio-laryngeal paralysis.

Such cases as those of Hun, Leyden, and others, as well as several which have come under my own experience, are, so far as their lower spinal phenomena are concerned, to be classed not with the protopathic, but the deuteropathic spinal amyotrophies of Charcot, the secondary spinal degenerations of Bouchard, to which attention will be given hereafter.

Even if we adopt M. Charcot's view that in such cases there is a real atrophy of the tongue, which is concealed by the hyperplasia of the perimysium, and the deposit of fat between the muscular fibres, we could not avoid perceiving the difference between such instances and those of true progressive muscular atrophy attacking the tongue, and in which there are fibrillations, and no interstitial fat to mask the veritable condition.

The lesions of the muscles in amyotrophic lateral spinal sclerosis are similar to those met with in progressive muscular atrophy. The perimysium is increased in quantity and the muscular fibrillæ undergo fatty degeneration and atrophy.

In considering the relation of the phenomena to the lesion, the questions to engage attention are mainly those which have already been sufficiently dwelt upon, when the other affections characterized by paralysis and atrophy were under notice. One symptom, spasmodic contraction, a concomitant of primary symmetrical spinal sclerosis, is, as has been pointed out when that disease was under consideration, the direct consequence of the lesion of the lateral columns.

---

[1] "Ueber progressive bulbäre Paralysie," *Archiv für Psychiatrie*, Band ii., S. iii.

Treatment.—In regard to a malady of so hopeless a character as amyotrophic lateral spinal sclerosis, there is little or nothing to say under this head. We have no means at our command capable of arresting the onward tendency of the disease.

VII.

INFLAMMATION (SCLEROSIS) OF THE POSTERIOR ROOT-ZONES OF THE SPINAL CORD (PROGRESSIVE LOCOMOTOR ATAXIA).

In the former editions of this work I described locomotor ataxia under the designation—based upon its patho-anatomy as then understood—of posterior spinal sclerosis. The recent investigations of Charcot and his pupils have, however, shown that the morbid process which gives rise to the remarkable group of symptoms known as locomotor ataxia, is in reality situated in the subdivisions of the posterior columns, lying between the columns of Goll and the posterior horns of gray matter, and called the posterior root-zones. In accordance, therefore, with its exact morbid anatomy, locomotor ataxia should be designated by the term placed at the head of this section. But, for convenience, I shall generally use the name locomotor ataxia, and no confusion can arise from this course, so long as we bear in mind the relation which it bears to the more exact pathological designation.

Although other writers, and especially Romberg,[1] had described a disease answering to that now generally known as locomotor ataxia, we are mainly indebted to Duchenne[2] for giving a full and distinct account of an affection which, before his studies, had scarcely attracted attention. Since then, the morbid anatomy, the pathology, and the symptomatology, have been so thoroughly studied by Charcot, Pierret, Westphal, and others, whose labors will be presently more specifically referred to, that the disease in question may be said, with truth, to be one of the most thoroughly understood in the whole range of medical science.

Symptoms.—Locomotor ataxia has no uniform set of initial symptoms. Sometimes it begins with dull, heavy pains in the small of the back or other part of the spinal column, which are very soon followed by sharp, electric-like pains, which shoot down the limbs along the course of the nerves, and which are very generally taken by the patient for twinges of neuralgia or rheumatism ; or it may be first manifested by a feeling of constriction around the body like that which is so common in acute myelitis.

---

[1] "Lehrbuch der Nervenkrankheiten," Berlin, 1840; also, "Sydenham Society's Translation," London, 1853.

[2] "De l'ataxie locomotrice progressive," *Archives Générales de Médecine*, 1858; also, "De l'électrisation localisée," Paris, 1861.

Again, the first symptoms are cerebral, and may consist of attacks of vertigo, pains either in the front or back of the head, epileptic fits, disturbances of vision, such as diplopia, ptosis, and defective accommodation. In this form the pupils are contracted often to mere points, or occasionally they will be found to be, one contracted, and the other dilated.

At other times the stomach and bowels are disordered ; vomiting is frequent, and there may be diarrhœa or obstinate constipation. Or, finally, the initial phenomena may be connected with the sensibility, giving rise to anæsthesia, and the various abnormal sensations connected therewith.

In whatever way it may begin, locomotor ataxia is soon chiefly manifested by disorders of motility, but inquiry reveals the fact that these are in reality secondary, being dependent upon the diminished sensibility which always exists. As this is the essential feature of the disease, I propose to inquire into its characteristics at some length.

If the lesion, as it usually does, exists in the dorso-lumbar region of the cord, the first evidences of anæsthesia or of perverted sensibility are noticed in the feet. A common feeling is as if the toes are too large for the shoe, or as if pieces of some plastic material are between them. Sometimes there are burning pains in the soles of the feet, and very generally "pins and needles" and other forms of numbness. A curious symptom is that, not only is the sensibility lessened, but the transmission of sensitive impressions to the brain does not take place with the normal degree of activity. I have noticed this phenomenon in rather more than half the cases that have come under my observation. In a lady, now a patient, a pin stuck into the calf of the leg is not felt for fourteen seconds on the right side and sixteen on the left. In a patient with posterior spinal sclerosis, under treatment in the New York State Hospital for Diseases of the Nervous System, if the feet were put into hot water the sensation was not felt for almost three minutes. As he said, "My feet might be scalded till the flesh dropped off and I would not know it till the mischief was done. Then I should feel it sharply." The explanation of this symptom is to be found in the fact that the conducting power of the posterior columns is lessened by the lesion, and hence the brain does not receive in the usual time the impressions made upon the nerves.

The ability to feel pain is therefore diminished, but there is, besides, a marked abatement of the tactile sensibility. The extent of this can only be accurately measured by the æsthesiometer. When this instrument is used, we find that the two points can be widely separated and a single impression only be felt on parts of the body which in the normal condition would give the sensation of two points at a much less distance apart. A gentleman from Virginia consulted me recently, in whom I diagnosticated locomotor ataxia, and who, instead of being able to perceive the two points with the end of the index-finger, when the twelfth of an inch apart, could feel but one point, though the two were separated to the extent of an inch and a half. Sometimes, even in the early stages of the disease, the loss of sensibility is so great that the patient hardly feels the points at all.

This loss of sensibility gives rise to some curious sensations, especially in the soles of the feet. These are usually such as might be produced by the interposition of some substance between the foot and the shoe, or between the shoe and the ground. One patient feels as if he has cushions on the soles of his feet, another as if bladders of air are interposed, another as if he is constantly treading on sticks, or, if riding in an omnibus, as if the hem of a lady's dress had got under his feet, and one a short time since described the sensation to me as being like that which he thought he would feel if his feet had been dipped into tar, and then into sand.

In some cases the ability to distinguish differences of temperature, or to appreciate the sensations produced by the application of hot or cold bodies to the skin of the affected parts, remains, but this is not, as some authors assert, a constant phenomenon, for in the majority of cases the sensations produced by heat or cold are just as unappreciable as those caused by any means capable of giving rise to sensitive impressions.

But the symptoms by which locomotor ataxia is recognized most readily are those which relate to motility, and the phenomena often make their appearance at a very early stage of the affection. At that time there is no loss of motor power, but there is an inability to coördinate the muscles—to bring them to harmonious action, and thus to execute with precision the various voluntary movements. Thus, in the act of standing, a great many muscles are simultaneously made to contract, and each one to just that necessary degree which is essential to maintain the body in the erect posture. Very often the first evidence of any motor trouble is experienced in regard to this faculty of standing. This impediment is, however, not one of paralysis, for, if the patient looks at his feet, he has no more trouble in standing alone than a perfectly sound man.

A gentleman connected with the city government of Brooklyn consulted me a short time since for an affection which was very evidently locomotor ataxia. The first indication of disease, as he informed me, was that it had been his habit, while at his morning ablutions, to shut his eyes, and he had noticed, about two months previously, that when he did so he could not maintain his equilibrium. When he visited me he was unable to stand with his eyes shut, and his gait was perfectly characteristic of locomotor ataxia.

Before the locomotion of the patient becomes obviously affected, he experiences inconvenience in placing his feet upon small surfaces. Thus, when he attempts to enter a carriage, he finds it difficult to guide his foot to the step, and in mounting a horse he cannot readily hit the stirrup. A gentleman from Maryland, who is now a patient of mine, and who is affected with the disease in question, tells me that among the first symptoms which he noticed was the difficulty he experienced in putting his foot into the stirrup. He was obliged to use his hand as a guide. A like trouble is frequently experienced in ascending a staircase.

The gait of a person suffering from locomotor ataxia is very much changed from that which is natural. Instead of the foot being placed upon the ground with an easy motion, the heel a little in advance of the sole, and the latter gliding down gently, the leg is, as it were, jerked forward, the heel comes down suddenly, and the sole follows, at a considerable interval, with an abrupt flapping motion. In ordinary walking the placing of the foot on the ground consists of one movement—there being no stoppage between the touching of the ground by the heel and the planting of the sole of the foot; but, in the gait of a person affected with posterior spinal sclerosis, the foot is placed on the ground by two distinct movements, one for the heel and another for the sole of the foot.

But, besides these irregularities of the progressive movements, there are others which are likewise notable. The leg is not carried directly forward, but is thrown out a little from the median line, and this gives the patient a motion like that of one walking on a tight-rope, and balancing himself with a pole. The object of this movement is doubtless to widen the base, and thus to enable the patient to preserve more readily his centre of gravity within it. In standing, he, for the same reason, always separates the feet to a greater than normal distance.

In walking or standing, it will be observed that the patient affected with sclerosis of the posterior root-zones of the spinal cord keeps his eyes fixed on his feet, or on the ground a little distance in advance. He is obliged to do this for the reasons—which with others will be more fully considered under the head of pathology—that the sensibility of the soles of the feet being diminished, and the muscular sensibility being also lessened, he is deprived, to a great extent, of the chief means by which he was formerly enabled to recognize the position of his feet, and of the dynamic condition of his muscles. He hence is obliged to make use of another sense—his vision—in order to obtain the necessary information. Therefore, when he shuts his eyes, or is obliged to walk in the dark, he is deprived of the assistance of his eyesight, and, having only his diminished tactile and muscular sensibility to guide him, moves in an exceedingly timid and disorderly manner, or else is unable to walk at all.

Under some circumstances he is unable to go forward, even with the assistance of his eyesight. Experience has taught him that he cannot rely on very important senses, which formerly he implicitly trusted. He loses confidence in them, and is not reassured, even with vision to assist him. He therefore uses extraordinary caution in walking over a tiled floor, on the ice or snow, in descending a staircase, or in crossing a street crowded with vehicles. In a recent clinical lecture,[1] delivered to the class of the Bellevue Hospital Medical College, I called special attention to this phenomenon of loss of confidence, and adduced several cases in illustration of this point.

That there is little paralysis of motion to account for these abnormalities, can be readily shown by a few inquiries and experiments. Thus it will ordinarily be found that the patient who is unable to stand with his eyes shut, or take a step in the dark, can push strongly with his legs, or walk a short distance with a good deal of vigor. He is still good for a "spurt," but long-continued muscular effort fatigues him.

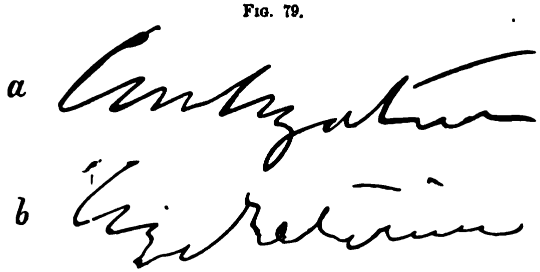
When the lesion is above the origin of the nerves which go to form the brachial plexus, the upper extremities are the seat of symptoms which are similar to those described as manifesting themselves in the legs. There are numbness and other indications of anæsthesia, together with more or less difficulty in coördinating the muscles into harmonious action. The patient finds that the ends of his fingers have lost, to some extent, their acute tactile sensibility, and that there is restraint in the management of the fingers. He experiences these difficulties in buttoning his clothes, in picking up a pin, in writing, and in other actions requiring nice manipulation. If he attempts, for instance, to carry a glass of wine to his lips, he spills a portion of the contents ; and, if told to place his finger on any particular part of his face, the movement is accomplished with a wabbling motion, and the finger is darted suddenly to the part as it approaches it. All persons possess a knowledge of where the different parts of their bodies are situated, which does not depend upon the sense of sight, although probably acquired by that sense and experience. There is such an intimate and exact relation between the ends of the fingers and the cutaneous surface of the body that, if a spot no bigger than the head of a pin be made with a pencil on the forehead, a person can close his eyes and touch it with the end of his finger without difficulty every time he makes the attempt. He can also, with the eyes shut, carry the end of his fingers straight to the tip of his ear, the middle of his eyebrow, or any other part of his body within reach. A person, however, laboring under sclerosis of the posterior root-zones of the spinal cord, cannot do any of these things. He loses, at a very early period of the disease, that intimate topographical relation which exists between the ends of the fingers and the rest of the body; and hence, when he closes his eyes, and attempts to put the tip of his index-finger on the end of his nose, he misses his aim, sometimes by as much as two or more inches.

---

[1] "Clinical Lectures on Diseases of the Nervous System," *Journal of Psychological Medicine*, January, 1871.

M. Onimus[1] has called attention to the fact that important indications are afforded by an examination of the handwriting of ataxics, the defective power of coördination being well shown even when the eyes are open, but being still more strongly manifested when they are shut. The difficulty which they experience is in making the round letters, such as *a*, *c*, and *o*. Besides the incoördination there is a jerking movement of the pen, and a kind of impulse to continue writing after the word is finished. Finally, when the ataxia of the arm is at its height, there is an impossibility of writing a single word, and we obtain only a set of traces confused and without order. I am able, after many experiments, to confirm the foregoing observations. In Fig. 79, *a*, is seen the attempt of a patient with his eyes open, and looking at his pen, to write the word "Civilization." At *b* is a like attempt made when the eyes were shut.

FIG. 79.



As in the legs, when the lesion is so low down in the cord as only to affect them, there is no well-marked paralysis. The grip of the patient is strong, and the dynamometer shows the existence of considerable strength. He is, however, not capable of continued muscular effort; and, though he may be able to lift several hundred pounds, or to carry another person around the room, his muscles are exhausted with the gradual and regular expenditure of a much less amount of force.

A phenomenon is often noticed as regards the upper extremities, which also exists with the lower, but which cannot be so readily manifested—and that is, that the patient loses the ability to distinguish even considerable differences between weights. In the normal condition, if two weights, differing in the ratio of thirty-nine to forty, are put one in one hand and one in the other, the difference is perceived without difficulty. The lower extremities, according to Jaccoud, are not so sensitive, and cannot distinguish a less difference than from about fifty to seventy grammes.

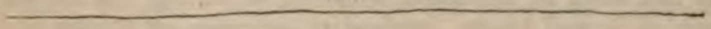
---

[1] *Gazette Médicale*, February 21, 1874; also, *Chicago Journal of Nervous and Mental Diseases*, April, 1874, p. 254.

A person affected with locomotor ataxia, due to sclerosis of the posterior root-zones above the origins of the nerves which form the brachial plexus, may have an ounce-weight put into his hand, and if in a few seconds it be removed, and one of half an ounce be substituted, he will not be able to tell correctly which is the heavier. Or both hands may be extended, and the two weights placed simultaneously in them. The eyes should, of course, be closed. Sometimes less differences can be perceived, but ordinarily greater ones are not distinguished. In the case of a gentleman now under my charge, there is an impossibility of telling which of two pieces of lead, the one weighing one ounce and the other a pound, is the heavier. Späth[1] states that, in a case under his charge, the patient could not distinguish between two weights, which differed as one to one hundred

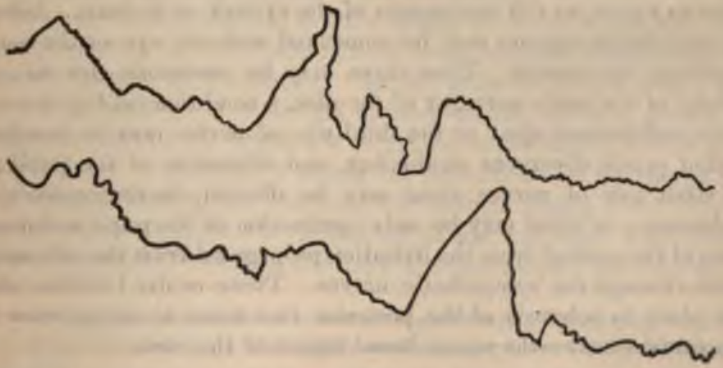
No means for measuring the extent to which the patient is able to determine the state of muscular contraction is at all comparable to the dynamograph. The range of its usefulness is, however, limited—owing to the fact that posterior spinal sclerosis is not very frequently seated high enough in the cord to affect the muscles of the upper extremities. When the lesion is not above the origin of the nerves which go to form the brachial plexus, the line is straight, as in the accompanying figure,

Fig. 80.



which represents the tracing made by a patient suffering from sclerosis of the posterior root-zones in the lower dorsal region of the cord. But, when the seat of the disease in the cord is anywhere between the fifth cervical and first dorsal vertebræ, the ability to maintain a uniform degree of pressure is impaired, and lines resembling the following are produced:

Fig. 81.



Both the above were made by the same patient, the upper with the right and the lower with the left hand. He was perfectly confident, till I showed him the tracings, that he had exerted a uniform pressure while the paper was traversing the pencil.

---

[1] "Beiträge zur Lehre von der Tabes dorsalis," Tübingen, 1864.

Under the name of baræsthesiometer, Eulenberg,[1] has recently described an instrument for estimating the sense of pressure, by means of which very accurate determinations can be made for different parts of the body. He succeeded in demonstrating a considerable impairment of the sense of weight in the great majority of cases of locomotor ataxia examined, even when sensibility to pain, tickling, or electric irritation, was but slightly affected, and the sense of temperature was normal.

The reflex excitability of the skin is generally notably increased. The touch of the bedclothes, or even the rubbing of one leg against the other, is sufficient to cause strong contractions. Involuntary movements of the limbs, independent of those due to reflex excitations, are rarely met with.

A symptom first pointed out by Westphal,[2] is the absence or notable diminution of the reflex excitability of the tendons. It is generally best exhibited by causing the patient to cross one leg over the other and then to strike with the side of the hand the tendon of the quadriceps extensor just above the patella. It will be found that there is very slight and often no movement whatever of the leg. In the healthy subject, as is well known, involuntary extension of the leg at once takes place. This symptom, though occasionally absent, as I have found, is yet so generally present, even in the early stage of locomotor ataxia, as to make it a sign of considerable diagnostic importance.

The electro-muscular contractility is generally increased. In some few instances I have found it normal, and in still fewer diminished.

It has already been mentioned that there are frequently ocular troubles. These generally occur among the early symptoms, and relate either to vision, to the movements of the eyeball, or to both. Indeed, the very first symptoms may be connected with the eye or the nerves supplying its muscles. Thus there may be amaurosis due to gray atrophy of the optic nerve, or of the disk, a condition readily detected by the ophthalmoscope ; or the third pair of nerves may be involved, causing ptosis, divergent strabismus, and dilatation of the pupil; or the sixth pair of nerves alone may be affected, causing convergent strabismus ; or there may be only contraction of the pupil and prominence of the eyeball from the irritation propagated from the cilio-spinal centre through the sympathetic nerves. These ocular troubles never take place in sclerosis of the posterior root-zones existing below the cilio-spinal centre—the upper dorsal region of the cord.

---

[1] *Allg. Med. Cent.-Zeitung*, No. 93, 1869; also, *Journal of Psychological Medicine*, October, 1870, p. 622.

[2] "Archiv für Psychiatrie und Nervenkrankheiten," B. v., s. 819.

Galezowski[1] has called attention to a very important fact in connection with the ocular disturbances of ataxics, and that is, the loss of the ability to distinguish certain tints and colors. Thus patients affected with locomotor ataxia, and who are at the same time amaurotic from gray atrophy of the optic nerves, are unable to distinguish the secondary tints of the scale (1 to 5, Plate F), and lose the perception of red and of green. The perception of yellow and blue is not lessened; on the contrary, it appears in some cases to be rendered abnormally delicate. I have frequently verified the extreme value of these tests, and have often observed the phenomena referred to when there was no other disturbance of normal vision, so far as all type-tests were concerned.

Another organ liable to functional derangement and even organic disease as effects of locomotor ataxia is the heart. Attention was first directed to this point by Berger and Rosenbach,[2] who, in a monograph based on seven cases, arrived at the conclusion that aortic insufficiency was the condition induced. But in a recent paper on the subject of the relation between locomotor ataxia and cardiac lesions, M. Grasset[3] shows, from two cases occurring in his own experience, and the citation of fifteen others from different writers, that the influence is not such a restricted one as supposed by Berger and Rosenbach, and that the influence is such as would be produced by acute suffering of any kind. He shows very conclusively that there is no direct relation between the spinal affection and the heart, but that the agonizing pains which the patient affected with locomotor ataxia usually suffers, are the cause of the heart-troubles. To use his own language: "Experiments prove the undoubted influence of peripheral excitations and painful sensations on the heart. They show that it is *possible* that, with man, pains, if long continued, affect the heart in an abnormal way and induce disease of the organ. Physiology simply indicates the *possibility;* clinical experience establishes the *reality*."

The disturbances in the healthy action of the stomach and intestines, which have already been alluded to as common initial symptoms, are sometimes very distressing. As the pains in the limbs are often taken for evidences of neuralgia or rheumatism, so these gastric and intestinal troubles are frequently regarded as indicating the existence of dyspepsia. I have had a number of patients under my charge who, with double vision, ptosis, contracted or unequal pupils, incoördination, and the other symptoms of locomotor ataxia, had been told that "it was all dyspepsia," because vomiting and gastric pain were prominent features of the disease. These symptoms are also due to the relations of the sympathetic nerves with the spinal cord, and are not present in cases where the lesion is low down in the lumbar region.

---

[1] "Du diagnostic des maladies des yeux par la chromatoscopie rétinienne," etc., Paris, 1868; also, "Échelles typographiques et chromatiques pour l'examen de l'acuité visuelle," Paris, 1874.

[2] "Ueber die Coincidenz von Tabes Dorsalis und Insufficienz der Aorten-Klappen," *Berliner klin. Wochenschrift*, No. 27, 1879, p. 402.

[3] "Ataxie locomotrice et les lésions cardiaques," *Montpellier Médical*, Juin, 1880.

When, however, this part of the cord is involved, there are very remarkable disorders of the genital system. These consist of frequent nocturnal emissions with or without erections, or of an inordinate desire for sexual intercourse. A gentleman who consulted me a few weeks ago, and who was affected with the disease in question, informed me that he had several times had as many as eight seminal emissions in one night, and that his sexual desire was almost inextinguishable.

Paralysis of the bladder is a common circumstance, and the sphincter is not infrequently likewise affected. The bowels are usually obstinately constipated.

The feeling of constriction around the body, which is so common a symptom in acute myelitis, and which is met with in other organic affections of the cord, is rarely absent in cases of sclerosis of the posterior columns.

Although the course of the disease in the great majority of cases is onward to a fatal termination, there are often periods of remission, as in other spinal affections, and it rarely happens that the duration is not several years. A gentleman from Westchester County, in this State, has been affected for over twenty years, and still walks tolerably well. Another from Boston had been subject to the disease for over twelve years. When I first saw him he could not stand with his eyes shut, had the characteristic ataxic gait, was subject to genital and urinary troubles, but yet was no worse than he had been six years previously. He visited me again in October, 1875, walking as well as when I saw him originally, but still subject to the electric-like pains in as great degree as ever. Another, from Pittsburg, has been in a stationary condition for several years; and another, from Binghamton, in this State, remains about as he was three years ago. I could easily cite twenty others whom I occasionally see professionally, who hold their own, and who have been affected for from five to ten years. Romberg gives the average duration at from ten to fifteen, Jaccoud at from six to eight, and all authors agree that the course is slow. Of the many patients affected with sclerosis of the posterior columns of the spinal cord who have been under my charge during the last ten years, five only have as yet died, so far as I am aware. Of these, one had been affected seven years, one eight years, two about ten years, and one eight and a half years. There are several cases now under my charge in which the affection has existed longer than either of these terms.

The advance of the disease in the cord causes an aggravation of all the symptoms, and the appearance of others not previously noticed. The loss of motor power is now a prominent feature, the muscles become atrophied, bed-sores make their appearance, there is anasarca, and the patient, if not carried off by some intercurrent affection, dies of the extreme exhaustion induced by his disease.

Among the anomalies of sclerosis of the posterior root-zones of the spinal cord, the joint affections are especially worthy of attention. Their connection with posterior spinal sclerosis was first indicated by Charcot.[1] Previous to his observations, they had been noticed, but they were ascribed to an intercurrent rheumatism, and, many years before locomotor ataxia was recognized as an independent disease, the association of spinal disease with inflammation of the joints was pointed out by Prof. J. K. Mitchell,[2] of Philadelphia; and his son, Dr. S. Weir Mitchell, with Drs. Morehouse and Keen,[3] had also related cases in which wounds of the spine had been followed by arthritis. Since Charcot's paper was published, Dr. Benjamin Ball[4] has cited cases of like affections coexisting with locomotor ataxia. In the cases in question there is no fever, redness, or pain. Generally these accidents disappear without leaving permanent organic changes behind them, but in some cases the head of the bone may be absorbed, and spontaneous dislocation be the result.

Of the cases of locomotor ataxia which have come under my observation, in nine only were there any troubles of the joints.

Death may take place either as the direct consequence of the lesion of the spine, or as the result of some intercurrent affection, such as pneumonia, dysentery, phthisis, or cystitis, or by disturbances of respiration and circulation, or paralysis of the muscles of deglutition by the extension of the disease upward, so as to reach the phrenic nerves, or medulla oblongata.

A psychical form of locomotor ataxia mentioned by some authors can scarcely be said to exist. It is true that some patients are peculiarly subject to mental depression, and to attacks of temporary excitement, with wakefulness; but the rule, according to my experience, is that by far the greater number preserve a very calm and equable frame of mind, and such is the conclusion of Steinhal[5] and Erb.[6]

But mental disorder of a very decided character is occasionally, though rarely, developed toward the latter stages of locomotor ataxia. I have not, however, been able to ascertain that this is particularly liable to assume any special form. It may be intense melancholia, or general mania, or general paralysis of the insane. It is necessary to bear in mind, especially as regards the last-named complication, that it is altogether different from a brain-disease with ataxic phenomena. There is a form of general paralysis of the insane in which there are difficulties of coördination and other tabetic symptoms, but here the cerebral manifestations are first in order; whereas, in locomotor ataxia, the spinal disorder is the primary trouble, and the cerebral altogether secondary. Westphal[1] was the first to direct attention to the ataxic form of general paralysis of the insane, and to show that the disorders of movement which are exhibited are due to degeneration of the posterior columns of the spinal cord.

---

[1] "Sur quelques arthropathies qui paraissent dépendre d'une lésion du cerveau ou de la moëlle épinière," *Archives de physiologie*, No. 1, January, 1868, p. 161.

[2] *American Journal of the Medical Sciences*, vol. viii., 1831, p. 55.

[3] "Gunshot-Wounds and other Injuries of Nerves," Philadelphia, 1864.

[4] "On Diseases of the Joints connected with Locomotor Ataxy," *Medical Times and Gazette*, October 31, 1868.

[5] "Beiträge zur Geschichte und Pathologie der Tabes Dorsalis," *Hufeland's Journal*, Band 9^{3}, 1844.

[6] "Graue Degeneration der Hinterstränge," *Ziemssen's Handbuch*, elfter Band, zweite Hälfte, p. 184.

Several instances of mental disorder supervening toward the termination of locomotor ataxia have come under my observation, and in one of them the development was so rapid as to preclude the idea that it was due to any extension of the disease directly to the brain. The form of mental derangement in this case was acute mania, and the patient died, after a paroxysm of intense excitement, in a condition of profound coma.

In another case there were repeated epileptiform convulsions, with stupor during the intervals, and in which latter condition death ensued.

Friedreich[2] has called attention to an affection of the spinal cord occurring very rarely in children, which he regards as a hereditary or family form of locomotor ataxia, but which, from an experience of four cases, and detailed descriptions of six others occurring in the practices of Drs. W. C. Warren, of Holly Springs, Mississippi, and E. S. Coleman, of Hollywood, Arkansas, I am disposed to think is not locomotor ataxia, but a hitherto unrecognized spinal disease. It begins in early life, and is, at least in the early stages, not so much characterized by incoördination as by muscular weakness. In the cases I have witnessed, the children, brothers in two instances, presented the appearance of old men, but were able to walk as well with the eyes shut as with them open, and to stand with closed eyes without any unusual swaying of the body. In none of the cases was there any hereditary tendency, but, as I have said, my cases are two pairs of brothers. Dr. Warren's cases, three in number, were children of the same parents; as were also Dr. Coleman's three cases. All my cases are males; of Dr. Warren's cases two were boys and one a girl; of Dr. Coleman's cases all were boys. Friedreich is of the opinion that this form is more common in girls than in boys. I think it somewhat doubtful whether the cases he cites are of the same character as those which have come to my knowledge, and I refrain from any further discussion of the subject till I have more thoroughly studied the symptoms and pathogeny, through the instances within the range of my own observation.

---

[1] "Tabes Dorsualis und Paralysie universal progressive," *Zeitschrift für Psychiatrie*, Band xx., 1863; und xxi., 1864.

[2] "Ueber ataxia mit besonderer Berücksichtigung der hereditären Formen," *Virchow's Archiv*, Band 68, 1876; Band 70, 1877.

Causes.—I have been very unsuccessful in my efforts to ascertain the cause in the greater number of persons affected with progressive locomotor ataxia who have been under my observation. The opinion is very prevalent that it is generally the result of excessive venereal indulgence ; and, although this is undoubtedly sometimes a cause, it certainly is not so common a one as is generally supposed. I have carefully inquired into the etiology of all the cases I have seen, and have only been able to assign inordinate sexual indulgence as the cause in a very small proportion. The impression has probably arisen from the fact that there are frequently aberrations of the sexual function as phenomena of the disease. Injuries and exposure to cold and dampness were apparently the causes in some cases, standing in a constrained position—three cases in railway conductors—in others, the excessive use of alcoholic liquors in a larger proportion, and syphilis in probably one-twentieth of the cases. In the majority, however, no cause can reasonably be assigned. As regards the predisposing causes, it is certainly more common in men than in women—four cases only in my experience pertaining to the female sex. The age from twenty-five to forty is that in which it most frequently appears. There seems to be no direct hereditary influence to the disease.

Diagnosis.—A consideration of the symptoms detailed in the foregoing pages will prevent posterior spinal sclerosis from being confounded with any other affection of the spinal cord. It may, however, be difficult at times to discriminate between it and the lesions of the cerebellum, and the distinction has frequently not been made by very skillful diagnosticians. At one time Duchenne held the view that locomotor ataxia was really the result of a lesion of the cerebellum, but he subsequently[1] retracted this opinion, and accepted the doctrine that the spinal cord is the seat of the disorder.

In a recent memoir[2] I have endeavored to point out the differences between cerebellar disease and the affection now called posterior spinal sclerosis. In that essay I have said : "Derangement of locomotion certainly does result from injury or disease of the cerebellum. Experimental physiology, as well as pathology, proves this. Beyond a doubt the disorder is, however, clearly due to vertigo. There are, moreover, headache, vomiting, and eventually in some cases hemiplegia, generally of the opposite side to that of the cerebellar lesion, a fact at variance with Larrey's assertion. The gait of a person thus affected is exactly similar to that of a drunken man. As Carre says, the movements are not abrupt, jerking, and exaggerated, as they are in locomotor ataxia. They are more uncertain, and do not depend upon any defect of co-ordination, but upon weakness of the voluntary power.

---

[1] "Diagnostic différential des affections cérébelleuses et de l'ataxie locomotrice progressive," *Gazette hebdomadaire*, 1866.

[2] "The Physiology and Pathology of the Cerebellum," *Journal of Psychological Medicine*, April, 1869.

"When either of the peduncles of the cerebellum is affected there is an irresistible impulse to go sideways, and sometimes gyratory movements are produced."

The characteristic symptom of cerebellar lesion is vertigo; and, although this is sometimes met with in sclerosis of the posterior root-zones, it is not a prominent feature, and is rarely present at all except in the very earliest stage.

In the cerebellar lesions the cutaneous sensibility is unimpaired, whereas in posterior spinal sclerosis it is always diminished.

A patient with disease of the cerebellum can stand and walk better with his eyes shut than with them open, for the vertigo is not in the former condition felt to the same extent. The reverse is true of locomotor ataxia. The history of the case will also serve as a good guide to the diagnosis. In the latter or even in the developed stage of locomotor ataxia it would be difficult to mistake it for any other affection.

Prognosis.—The prognosis is no more favorable than that of anterior or lateral spinal sclerosis. A few cases are cured, more are relieved, but the great majority go on unchecked. Of the cases which have come under my observation, seven were cured, and they were subjected to treatment from a very early stage. Of these, four were probably of syphilitic origin, but in the other three no such cause was at all probable. One of them was a woman.

The cases in which amelioration has been produced are more numerous. In fact, it is not at all uncommon to succeed in retarding the onward progress of the disease, and of thus prolonging the life of the patient.

Morbid Anatomy.—Within the last few years many very important contributions have been made to the morbid anatomy of locomotor ataxia mainly by the pathologists of that great French school of the Salpêtrière with Charcot at its head. For the complete understanding of these a few words relative to the normal anatomy of the parts concerned are necessary.

In embryonic and early infantile life the posterior columns are divided into two unequal parts by a fissure extending from the angle formed by the posterior median fissure and the posterior commissure of gray matter. The internal or median part is wedge-shaped, is of greater extent in the cervical region than in the dorsal, and greater in this than in the lumbar. It is called the slender column of Brudach, cuneiform column or column of Goll.

The external part of the posterior column is all that region bounded externally by the posterior horn of gray substance and interiorly by the column of Goll. It is called the external band of the posterior column, or the posterior root-zone.

In adult life the fissure separating these two regions no longer exists, but its situation is generally marked by a furrow on the periphery of the cord, and a histological difference exists between them, in that the columns of Goll contain a greater amount of connective tissue than do the posterior root-zones.

Now, although it often happens that both these subdivisions of the posterior columns are the seat of the alteration giving rise to locomotor ataxia, it has been very positively shown that the essential lesion is that of the posterior root-zones, and that it is to the disease of these regions that the peculiar symptoms of locomotor ataxia are due. In fact sclerosis of the posterior columns may exist and there be no ataxic symptoms whatever, as is shown by the secondary phenomena of Pott's disease. Here a degeneration or sclerosis of a part of the posterior columns throughout their whole extent exists, and there are none of the characteristic symptoms of locomotor ataxia present. But the apparent discrepancy is explained when we come to find that in these cases the lesions are confined entirely to the columns of Goll.

As we shall see hereafter, this is not the only evidence, for the columns of Goll are subject to primary sclerosis without the production of ataxic symptoms.

On the other hand, when the posterior root-zones are sclerosed, the phenomena of locomotor ataxia are always present, whether the columns of Goll be involved or not. This point has been determined by a case very thoroughly investigated by Pierret,[1] in which a woman named Moli suffered from the electric-like pains, and incoördination of locomotor ataxia, which were mainly experienced in the upper extremities. On post-mortem examination that part of the cord—the cervico-dorsal—in relation with the upper extremities was found to be sclerosed in a thin lamina existing only in the posterior root-zones. The columns of Goll were perfectly healthy.

In another case the same observer had the opportunity of confirming the results previously obtained. A woman (Cutta) had suffered for many years with electric-like pains in the lower extremities, plantar anæsthesia, and incoördination. Standing and walking were impossible. In late years she had experienced constricting pains around the body. The superior extremities were not in the least involved. On post-mortem examination the posterior columns in the lumbar region were sclerosed throughout their whole extent, except that on each side a little islet of healthy tissue remained. At the sixth dorsal vertebra the sclerosed tissue was less extensive and almost entirely confined to the columns of Goll, the posterior root-zones only exhibiting on each side a little islet of sclerosed tissue. A little higher these islets disappeared and the lesion was entirely limited to the columns of Goll.[1]

---

[1] "Sur les altérations de la substance grise de la moëlle épinière dans l'ataxie locomotrice," etc., *Archives de physiologie*, 1870, p. 597.

Now, if the lesion of the columns of Goll had been at all connected with the symptoms observed during life in the lower extremities, we should not be able to account for the entire exemption of the superior extremities from all ataxic phenomena, for the columns of Goll in that part of the cord in relation with them were the seat of marked lesion.

In another case reported in the same memoir, both upper and both lower extremities were affected, and on post-mortem examination the whole of the posterior columns in the lumbar, dorsal, and cervical region were altered.

Hence, comparing this case with the other, the conclusion is irresistibly arrived at that the ataxic phenomena exhibited in the upper extremities of this second case were due to the lesion of the posterior root-zones, and not to that of the columns of Goll.

Even when the columns of Goll are the seat of marked disease, it is more than probable that the lesion originates in the posterior root-zones and therefore affects the columns of Goll secondarily. Pierret expresses the opinion, in which Charcot concurs, that the implication of the columns of Goll is a phenomenon analogous to that which produces an ascending median sclerosis as a result of partial myelitis, and that the lesion is only produced in those cases in which the morbid process is very strongly pronounced in the lumbo-dorsal region of the cord.

In a subsequent memoir, M. Pierret[2] discusses with great ability the anatomical disposition of the fibres entering into the composition of the posterior columns of the cord, and shows that the external fasciculi are in reality the posterior root-zones, and that it is in them the essential lesion of locomotor ataxia resides.

But it is not always the case that the morbid process stops with the posterior root-zones and the columns of Goll; the posterior horns of gray matter, the lateral columns, and even the anterior horns, may be reached.

As to the spinal nerves, it will almost invariably be found that the posterior roots are atrophied.

The intra-cranial lesions are important. Indeed, there is reason to think that they are often the starting-point of the disease. They have been very carefully studied by many observers, and the fact that one of the most striking of them—that of the optic nerve—can be observed with the ophthalmoscope, gives additional interest to the subject.

The alteration which the optic nerves undergo is a slow progressive sclerosis, causing atrophy of the disks and of the nerves themselves. From the color which the nerves assume, the condition is known by ophthalmologists as gray degeneration. According to Leber, and Wecker and Jaeger, the essential changes met with in gray degeneration of the optic nerve are a marked increase in the quantity of connective tissue, especially of the cell-elements, and the appearance of numerous grumous cells. The lesion is therefore of the same character as sclerosis affecting the other parts of the nervous system.

---

[1] "Note sur la sclérose des cordons postérieurs dans l'ataxie locomotrice progressive," *Archives de physiologie*, tome iv., 1871–'72, p. 364.

[2] "Considérations anatomiques et pathologiques sur le faisceau postérieur de la moëlle épinière," *Archives de physiologie*, 1873, p. 334.

The ophthalmoscopic appearances have been so clearly stated by Wecker and Jaeger,[1] that I quote from them the following details:

"The clinical characters are especially revealed by the particular appearance of the papilla and by the narrowing of the visual field.

"An essential sign which we have claimed for the ophthalmoscopic image of gray degeneration, is the more or less complete absence of an atrophic excavation. It is of course easy to understand that such excavation is much less apt to be formed when there is a substitution of cellular tissue than when, as in simple atrophy, the entire nervous structure disappears.

"In gray degeneration of the nerve the initial signs of the disease consist in a simple change in the color of the papilla without any excavation. It becomes pale, as is perceived by the examination of the erect image with Helmholtz's plates, and it assumes a more or less pronounced bluish tint.

"With this change of color there is a coincident change in the transparency of the tissue of the disk. It becomes impossible to follow the central vessels in their ramifications; they seem to be applied to the bluish-white tissue of the papilla, and the whitish sclerotic ring offers a marked contrast to the opaque tissue of the nerve."

According to these authors, the ophthalmoscopic appearances in cases of gray degeneration are sufficiently characteristic to enable the diagnosis of locomotor ataxia to be made with certainty from them alone. This is, however, I am inclined to think, too positive a statement. We may, however, safely conclude that when they are coexistent with the disturbance of chromatic perception previously referred to; when the pupils are contracted—they are dilated in ordinary optic neuritis and atrophy of the optic nerve—and especially when electric-like pains are present, we have as positive indications of the existence of locomotor ataxia as are desirable.

Besides this atrophy of the optic nerve, there is another condition to which it is subject, as a consequence of a preëxisting sclerosis of the posterior root-zones, and that is a chronic neuritis. This state is induced when the spinal lesion is seated in that part of the cord known as the cilio-spinal centre. The ophthalmoscope in these cases reveals in the early stages the existence of choked disk, and subsequently simple atrophic changes. This condition is not peculiar to locomotor ataxia, but may be caused by other chronic affections of the spinal cord. It is referred to by Dr. Clifford Allbutt[1] in his excellent monograph as "simple or primary atrophy of the optic nerve, sometimes accompanied at first by that slight hyperæmia and inactive proliferation which make up the state I have called chronic neuritis. This sort of change I have never found as a result of spinal injuries, but I have often met with it in chronic degeneration of the cord, and in locomotor ataxy."

---

[1] "Traité des maladies du fond de l'œil," Paris, 1870, p. 73.

Besides the optic, others of the cerebral nerves may be affected. Those most commonly involved are the third, the sixth, and the auditory; the lesion of this latter causing deafness and other disturbances of hearing.

The lesions found in the brain never affect primarily the hemispheres. To be sure, it is sometimes the case that there are mental troubles, but they come on toward the close, and are probably the result of defective nutrition and sympathetic action.

The other cerebral lesions, like that of the optic nerve, are in very intimate anatomical relation with the posterior columns of the cord. They are, therefore, met with in the lower cerebellar peduncles, in the restiform bodies, and in the optic thalami, and consist of degeneration and atrophy.

The situations of the spinal lesions and their general character were well known to Romberg[2] before the researches of Duchenne, Charcot, and others. Thus, he states that he was present at the post-mortem examination of the cord of a former patient. The organ was reduced one-third in diameter, and the atrophy was confined to the lower part of the posterior columns. The posterior nerve-roots were also atrophied, but the anterior columns were healthy. He was also acquainted with the fact that the cerebral nerves were similarly affected.

Although it is probable that the sympathetic is atrophied in some part of its extent, in many cases of locomotor ataxia, the fact has not been demonstrated, except as regards one instance reported by Donnezan, in which a filament from the superior cervical ganglion was found atrophied. The ganglion itself was healthy.

In the later stages of the affection the muscles may exhibit a condition of atrophy. In such cases their tissue will be found on microscopical examination to have undergone fatty degeneration and substitution to a greater or less extent.

---

[1] "On the Use of the Ophthalmoscope in Diseases of the Nervous System," etc., London and New York, 1871, p. 198.

[2] "Lehrbuch der Nervenkrankheiten des Menschen," "Sydenham Society Translation," London, 1853, vol. ii., p. 399.

The morbid anatomy of the joint-affections which sometimes result from the spinal lesion consists in an accumulation of water in the synovial cavity, and a general œdema of the soft parts. The most common seat of this alteration is the knee, and next after that the shoulder. The hip, the elbow, the wrist, and the smaller joints, may also be involved. Occasionally, the trouble does not stop here, but the articulating surfaces may become rough from atrophy of the proper bone-tissue, and eventually a considerable part of the osseous substance disappears, giving rise to spontaneous laxation. The accompanying figures, from Charcot, illustrate the nature of the change. In Fig. 82 is represented the superior extremity of the healthy humerus, and in Fig. 83 the corresponding part of a humerus exhibiting the lesions produced by locomotor ataxia.

FIG. 82.

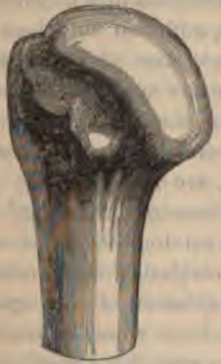
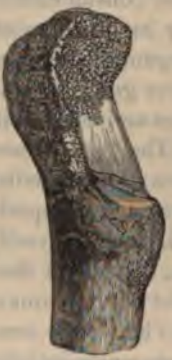


FIG. 83.



Pathology.—The theory of posterior spinal sclerosis which is generally held is, that the lesion impairs a faculty by which the muscles are brought into harmonious action—a faculty of coördination. According to this view, the first thing to be done was to locate this faculty in an organ, and Duchenne, with whom it originated, adopting the ideas of Flourens and others, placed it in the cerebellum, and therefore regarded what he designated progressive locomotor ataxia as a disease of the cerebellum.[1] Thus he said:

"In conclusion, regarding the order of appearance, and the habitual progress of the symptoms which mark the three periods of progressive locomotor ataxia, we find that the central morbid action which produces the phenomena symptomatic of this disease begins in general in the motor nerves of the eye, and in the tubercular quadrigemina, extending thence to the superior and inferior cerebellar peduncles and finally to the cerebellum."

As already stated, Duchenne has since abandoned this view of the location, and now assigns its seat to the posterior columns of the cord, but, in order to make the morbid anatomy agree with the theory of the disease which he holds, he places his faculty of coördination in the cord. But, although it has been established by numerous post-mortem examinations that the cerebellum is not the seat of lesion in cases of locomotor ataxia, and although the differential diagnosis between diseases of the cerebellum and posterior spinal sclerosis has been very clearly made out, there are some who still hold the view that, although the cerebellum shows no traces of disease, and that, although the posterior columns of the spinal cord may be in a state of sclerosis, the symptoms are the result of an interruption to the passage, from the cerebellum through the posterior columns to the spinal nerves, of that force which coördinates the muscles into harmonious action. In the memoir to which reference has already been made, I have entered at length into the consideration of the question of the location of a coördinating faculty in the cerebellum, and have, I think, adduced sufficient facts and arguments to show that coördination is not one of its functions. Without going into a full account of the subject, a synopsis of the conclusions arrived at will probably not be deemed out of place:

---

[1] "De l'électrisation localisée," deuxième édition, Paris, 1861, p. 611.

1. The consequences of removal of the cerebellum, if the animal survives the immediate effects of the injury, are not enduring. This conclusion is supported by experiments by Flourens,[1] Harting,[2] Wagner,[3] Dalton,[4] myself,[5] and others. The physiological inference, of course, is, that, if the faculty of coördination resided in the cerebellum, it ought to be permanently removed with the ablation of the organ.

2. The entire removal of the cerebellum from some animals does not apparently interfere in the slightest degree even for a moment with the regularity and order of their movements. I have performed a number of experiments with reference to this point, on different classes of animals. They prove very clearly that the cerebellum is not the generator of coördinating power in all animals that have it: a fact in comparative physiology which is fatal to the hypothesis that this is its function in man.

3. The disorder of movements which results in birds and mammals immediately after injury of the cerebellum is not due to any loss of coördinating power, but is the result of vertigo.

If the cerebellum be removed from a pigeon it exhibits disorder in its movements, but a careful examination of the phenomena exhibited, shows that it is suffering from a vertiginous sensation. Even when placed upon its breast and allowed to remain at rest, there is a trembling and swaying of the body, such as is produced by alcoholic intoxication. Exactly such symptoms can be caused by giving pigeons bread soaked in alcohol.

---

[1] "Recherches expérimentales sur les propriétés et les fouctions du système nerveux," Paris, 1842.

[2] "Experimenta quædam de affectibus læsionum in partibus encephale," 1826.

[3] "Nachrichten von der Universität und der Königl. Gesellschaft der Wissenschaften zu Göttingen;" also, *Journal de la physiologie de l'homme et des animaux*, Avril, 1861.

[4] *American Journal of the Medical Sciences*, January, 1861, p. 83; also, "Treatise on Human Physiology," fourth edition, 1867, p. 416.

[5] *Op. cit.*, p. 24.

4. The phenomena of cerebellar disease or injury, as exhibited in man, are not such as show any derangement of the coördinating power.

Many cases are on record which support this proposition. Andral[1] states that, of ninety-three cases of cerebellar disease which he has studied, only one appeared to support the theory which locates the coordinating power in the cerebellum.

Many special instances might be brought forward, and several have occurred in my own practice. The case of Alexandrine Labrosse, reported by Combette,[2] is, however, worth referring to more specifically. His paper is entitled "Case of a young girl who died in her eleventh year, in whom there was complete absence of the cerebellum, of the posterior peduncles, and of the annular protuberance." Magendie examined the brain after her death, and satisfied himself that the defect was congenital. As M. Combette remarks in regard to this case, Alexandrine Labrosse had been able to walk for several years, but always in an uncertain manner. Gradually her legs lost their strength, and she became paraplegic. She preserved the use of her upper extremities to the last. It is very evident, therefore, that the weakness of her legs was due to paralysis, for, had it been the result of incoördination, the arms must necessarily have participated.

For these reasons, I think, it cannot be considered, with any degree of probability, that the cerebellum has any thing whatever to do with the symptoms manifested in sclerosis of the posterior root-zones of the cord. Neither is it, in my opinion, necessary to assume the existence of an organ whose office it is to exercise a coördinating power.

Other authors have ascribed the incoördination which is so prominent a phenomenon of sclerosis of the posterior root-zones to the loss of what they call the muscular sense.

Sir Charles Bell[3] has argued strongly in support of the existence of such a sense. He enunciates his theory in the following sentence:

"Between the brain and the muscles there is a circle of nerves; one nerve conveys the influence from the brain to the muscle, another gives the sense of the condition of the muscle to the brain."

It is by this connection that we are enabled, according to Sir Charles

---

[1] "Clinique médicale," seconde édition, tome v., p. 735.

[2] *Journal de physiologie expérimentale et pathologique*, par F. Magendie, tome xi., Paris, 1831, p. 27.

[3] "On the Nervous Circle which connects the Voluntary Muscles with the Brain," "Philosophical Transactions." Also, "The Nervous System of the Human Body," London, 1830, p. 225.

Bell and other physiologists, to form an idea of the state of contraction of a muscle, and to lessen or increase the contraction as may be necessary. According to some writers, in locomotor ataxia the patient loses this muscular sense, or is unable to exert it, for the reason that the spinal columns through which the perception reaches the brain are, by disease, rendered incapable of transmitting it.

In my opinion—and I shall endeavor to support it presently—there is no such perception as that referred to, and its existence is certainly not established by the case reported by Dr. Ley to Sir Charles Bell, and which is incorrectly quoted by Trousseau; for it proves nothing more than that defective sensibility existed, and that the sense of sight had to be used in order to obtain a correct idea of what the insensible muscles were doing.

A lady having been recently delivered, and having suffered severe hæmorrhage, was seized soon afterward with headache and numbness. Dr. Ley was called to see her.

"I found her," he says, "laboring under severe headache, not confined to, but infinitely more violent upon one side than the other, and occupying the region of the temporal and occipital bones above the mastoid process, and attended with considerable pulsation.

"Upon one side of the body there was such defective sensibility, without, however, corresponding diminution of power in the muscles of volition, that she could hold her child in the arm of that side so long as her attention was directed to it; but, if surrounding objects withdrew her from the notice of the state of her arm, the flexors gradually relaxed, and the child was in hazard of falling. The breast, too, upon that side, partook of the insensibility, although the secretion of milk was as copious as in the other. She could see the child sucking and swallowing, but she had no consciousness from feeling that the child was so occupied. Turgescence of that breast produced no suffering, and she was unconscious of what is termed the *draught* on that side, although that sensation was strongly marked in the other breast.

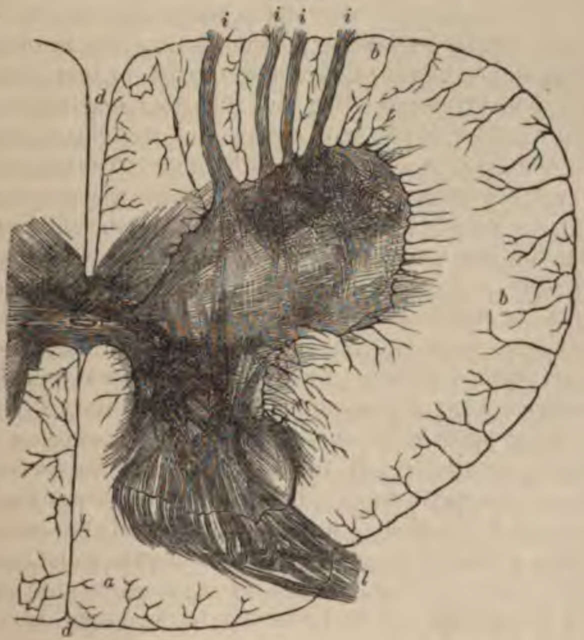
"Upon the opposite side of the body there was defective power of motion, without, however, any diminution of sensibility. The arm was incapable of supporting the child, the hand was powerless in its grip, and the leg was moved with difficulty and with the ordinary rotary movement of a paralytic patient, but the power of sensation was so far from being impaired that she constantly complained of an uncomfortable sense of heat, a painful tingling, and more than the usual degree of uneasiness from pressure or other modes of slight mechanical violence."

After a few months she died; having, in the mean time, received no improvement from the active treatment employed, and having also become pregnant again. On post-mortem examination there were found evidences of chronic inflammation of the membranes of the brain. The cord was not examined. Certainly this case presents nothing which may not be met with in any patient who has anæsthesia on one side and paralysis of motion on the other. I have observed a number of similar cases, and they neither prove the existence of a muscular sense, nor do they have any special bearing on posterior spinal sclerosis beyond the fact that they exhibit deficient sensibility.

But, before proceeding to the further discussion of this subject, clear ideas should be entertained relative to the anatomy and physiology of the spinal cord. The researches of Dr. J. Lockhart Clarke have given us very exact information on these points, and I shall therefore quote from him in full.[1]

As Dr. Clarke states, before he began his researches on the structure of the spinal cord, it was universally taught, both in England and abroad, that the posterior roots of the spinal nerves were attached exclusively to the lateral columns of the cord; whereas he showed, what is now universally admitted, that they are attached immediately to the posterior columns, and not at all to the lateral. The importance of this fact in both a physiological and pathological point of view, and especially in its relation to sclerosis of the posterior root-zones, will presently appear.

FIG. 84.

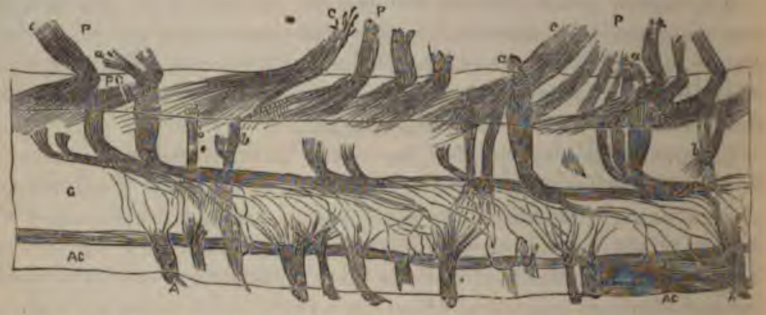


In Fig. 84, which represents a transverse section of the right lateral half of the lumbar enlargement of the cord, the posterior nerve-roots (*l*) are seen to enter through nearly the entire breadth of the posterior column, excepting that strip nearest the posterior median fissure called the column of Goll (*a*); and in Fig. 85, which represents a longitudinal section of the cervical enlargement of the cord, we see the course of the roots of four consecutive nerves (P, P, P, P) within the cord. These roots are of three kinds : The first kind (*a*, *a*, *a*, *a*) enter the cord transversely, and pursue a very remarkable course. Each bundle, after traversing the longitudinal fibres of the posterior column (PC) in a compact form, and at a right angle, continues in the same direction to a considerable but variable depth within the gray substance (G), dilating and again contracting, so as to assume a fusiform appearance. It there bends round upon itself, at a right or more obtuse angle, and, running for a considerable distance in a longitudinal direction down the middle of the cord, sends forward, at short intervals, into the anterior gray substance, a series of fibres, some of which mingle with those of the anterior roots (A), while others enter the anterior white column, as at AC, AC, in which they run longitudinally, both upward and downward.

---

[1] *See* Dr. Clarke's paper on "Locomotor Ataxy," in *British Medical Journal*, September 25, 1869, p. 344, from which I take this account and the accompanying woodcuts.

FIG. 85.



The second kind of posterior roots (*b*, *b*, *b*) also traverse the posterior column transversely, but sometimes a little obliquely from without inward. Their component fibres are finer than those of the other bundles, measuring about the $\frac{1}{7000}$th of an inch in diameter. Some of these fibres cross over transversely to the gray substance of the opposite side through the posterior commissure behind the canal. Others extend into the posterior and lateral white columns of the same side, while the rest may be traced deeply into the anterior gray substance (G, Fig. 85), where they diverge in different directions, and are ultimately lost to view.

The bundles forming the third kind of posterior roots (*c*, *c*, *c*, Fig. 85) enter the end obliquely. A few of their fibres proceed near the surface of the posterior column both upward and downward, and pass out again with roots above and below them. The rest cross the posterior column obliquely and chiefly upward, a small number only running downward. Interlacing at the same time with each other and with the roots already described, these fibres diverge, and for the most part reach the gray substance at points successively more distant from their entrance into the cord in proportion to the obliquity of their course, the most divergent and superficial taking a longitudinal course at least for some distance, with the fibres of the posterior column, among which they are lost. From these investigations ("Philosophical Transactions," 1853), Dr. Clarke inferred that the posterior white columns of the cord cannot be the only channels for the transmission of sensory impressions, an inference which was verified two years later by the experiments of Brown-Séquard (*Gazette médicale*, 1855).

Such being the anatomical connection of the posterior nerve-roots with the posterior columns of the cord, it is evident that no part of the length of those columns except the fasciculi bordering on the posterior median fissure—the columns of Goll—can be damaged either by injury or disease without involving destruction of a corresponding number of nerve-roots; and, since reflex action of the cord requires that impressions be conveyed by nerve-roots to the gray substance, the diminution of reflex action in cases of injury to the posterior columns is thus readily explained.

We have also in these facts an explanation of the pains which are felt in the parts supplied by nerves coming from the diseased parts of the cord. The sclerotic process causes pressure to be exerted on the roots, and, in accordance with the law that irritation applied to a nerve-centre or trunk is felt most acutely in the peripheral branches, we have the pain experienced not in the cord but in distant regions.

Subsequently, as the integrity of the nerve-roots and of the parenchymatous substance of the posterior root-zones becomes destroyed, the hyperæsthesia is succeeded by anæsthesia, and indeed in many cases coexists with it from the very earliest development of the symptoms.

Dr. Clarke, in the anatomy and physiology of the posterior nerve-roots and their relations to the cord, which he has thus so satisfactorily elucidated, presents a theory of the phenomena of incoördination met with in posterior spinal sclerosis, to which I will allude more specifically directly. In the mean time a few words in reference to the "muscular sense" are necessary to the understanding of the whole subject.

Landry[1] declares that, whenever a muscle is caused to contract, the brain perceives the seat and the extent of the contraction. I am very sure that no sensation starts from the muscle which can give the brain any idea on the subject. As Trousseau[2] remarks:

---

[1] "Mémoire sur la paralysie du sentiment d'activité musculaire," Paris, 1855.

[2] *Op. cit.*, p. 159.

"An important distinction must be drawn between the consciousness of a movement which has been executed, and the consciousness of the muscular contraction which performs the movement. When after shutting our eyes we execute without effort a pretty extensive movement, we are unable, even on paying the strictest attention, to feel the contraction of our muscles, although we may feel the movement communicated to the lever by the contracted muscles. This fact is so true, that, when we ask an intelligent person, who knows nothing of anatomy and physiology, which is the seat of the movements through which the fingers are flexed or extended, he immediately points to the hand, and never to the forearm. It is only when the muscular effect is considerable and kept up for a long time, that it is perceived where the contraction really occurs. Normally, then, we have no consciousness of muscular activity, but merely the consciousness of the movement itself, which is a perfectly different thing."

In a very thorough essay on the subject, Dr. Bastian[1] has discussed the whole subject of the "muscular sense." He denies—and I think with good reason—the existence of any such special sense. In his opinion, there is no consciousness of the state of muscular contraction, and that the estimations by which we regulate the extent to which it is necessary, for instance, to contract the muscles of the upper extremity to sustain a certain weight in the hand, are "inferences based upon previous sensory impressions of the passive kind, upon impressions emanating from the skin, from the joints, and from the muscles themselves, so that in my opinion there are *no* conscious impressions derivable through the 'muscular sense.'" This, as I think, is not to be considered as an appanage of the intellect, but rather as an unconscious organic guide in the performance of voluntary movements. Why, it may be asked, do I not, as Trousseau has already done, deny its existence altogether? And to this I should reply by saying: "Although there is no evidence to lead us to believe that we derive any conscious impressions through the intervention of this so-called 'muscular sense,' there *is* evidence to show that the brain is assisted in the execution of voluntary movements by guiding impressions of some kind, which, while they differ from the impressions producible by means of the ordinary cutaneous and deep sensibility, may differ still further from these, owing to the fact of their not being revealed in consciousness at all."

This impresses me as being a very philosophical view of this rather difficult question, and it is in part sufficient to explain the incoördination existing in cases of sclerosis of the posterior root-zones; but, for the full understanding of the subject, it appears to me we must bring forward another fact in the physiology of the spinal cord which has not hitherto, so far as I know, been made applicable. The spinal cord serves two distinct purposes in the economy. It transmits nervous force to and from the brain, and it is a centre which generates nervous force. Referring now to the anatomical details given by Dr. Lockhart Clarke, we find that the posterior nerve-roots not only reach the white substance of the posterior root-zones and antero-lateral columns, but that they are in intimate relation with the gray matter. Now, the white substance simply serves for the transmission of nervous force, the gray elaborates it. Hence a great many of the muscular actions which we perform are done through the agency of this gray matter of the cord, and are without the intervention of the brain, and the brain can only be brought to bear upon them through the agency of the white substance. The states of muscular contraction are, therefore, in all probability, perceived by the gray substance of the cord, and, as the brain has no consciousness of the perceptions of the cord, we are not made aware of the states of muscular contraction. The muscular sense, therefore, does not exist, at least in the same manner as do the other senses.

---

[1] Remarks on the "Muscular Sense" and on the "Physiology of Thinking," *British Medical Journal*, May 1, 1869, and subsequent numbers.

In sclerosis of the posterior root-zones of the spinal cord the lesion generally involves the posterior nerve-roots, the posterior white substance, and the posterior cornua of gray substance. Hence the cord loses both in the ability to transmit and to generate nervous force. Those unconscious acts of muscular coördination which are regulated by the gray substance of the spinal cord can no longer be perfectly accomplished, and the brain is brought to assist in the determination through the sense of sight. The patient cannot stand well with his eyes shut, or walk in the dark, or determine differences of weight, because he is relying altogether on the perceptive faculty of the spinal cord, and this organ is not in a condition to perform its work with precision; and his movements and muscular contractions are rendered still more uncertain from the fact that the cutaneous sensibility is diminished.

In the normal condition we frequently—in determining weights, for instance—are greatly assisted by the sense of sight, and there is nothing surprising in the fact that, in a disease like locomotor ataxia, the spinal cord should be unable to perceive states of muscular contraction without the assistance of the brain. And, as the conducting power of the cord is also lessened, the brain cannot act with its full power; and, therefore, even with all the assistance to be derived from the chief generator of nervous force in the body, the patient's muscles are not so well coördinated as in health.

Dr. Lockhart Clarke, in the memoir already cited, explains the incoordination upon another principle, which, although, as I think, not sufficient to account for the phenomenon, may, and probably does, exercise some influence. His view is, that there is a physiological state of the muscles dependent on reflex action, that is absolutely essential to the proper coördination of voluntary movements, and that is, their tonicity, or that moderate but constant state of contraction which keeps the antagonist muscles, or those that are variously opposed to each other, in equilibrium or static tension. In the performance of voluntary movements a constantly-varying number of muscles, each of which differs more or less in force and in the particular direction which it gives to the limb or part, are associated together in action in an endless variety of ways. Each of the muscles that compose these varying groups must contract either simultaneously or successively to a certain particular extent, with a certain degree of force, and with a certain degree of rapidity, in relation to the actions of the others, according to the resultant direction desired in the voluntary effort; and this endless variety of ways, in which a constantly-varying number of muscles are balanced against each other in contraction for the performance of constantly-varying and complicated voluntary movements, affords the most exquisite and beautiful example of what, in physical science, is termed the composition of forces. In this balancing of muscular force we have to learn by experience, and to remember the exact voluntary effort required to contract each muscle to its proper extent, with its proper force, and with its proper degree of rapidity, in relation to the action of the others that complete the group employed. Now, it is evident that if some of the muscles of the group employed have lost their normal tension or tone, they will not properly respond to the excitation of the voluntary stimulus, and will fail to perform their proper part in balancing the effects of the other muscles of the group that retain their tension, in the execution of any given movement. In proportion, therefore, to the exact amount of tension lost by any muscle or muscles of the group, and the number of muscles that have lost that tension, there must necessarily be a proportionate amount of disorderly movement of incoördination. But it appears to be satisfactorily proved, by the experiments of Brondigeest, Rosenthal, and others, that this constant tension or tone of the voluntary muscles is due to a constant reflex action of the cerebro-spinal centres, and is immediately dependent on impressions conveyed from the muscles to those centres by the posterior roots of the nerves. Now, Dr. Clarke has shown how these posterior spinal roots are spread out through the posterior columns of the cord; how impossible it is for these columns to be destroyed to any great extent without involving destruction of the nerve-roots; and how, consequently, the columns are so destroyed in locomotor ataxia. But, except in the very last stage of this malady, all the posterior roots are not injured by disintegration, and some of them are still competent to carry impressions to the gray substance of the cord; so that some of the muscles retain their tone, while others lose it to a greater or less extent.

This is Dr. Clarke's explanation of the peculiar feature of posterior spinal sclerosis, in almost his own words. That it is ingenious and plausible, all physiologists and pathologists will admit.

An interesting point connected with the pathology of locomotor ataxia is the fact that the spinal lesions sometimes exist in conjunction with the cerebral lesions which are the anatomical basis of general paralysis of the insane. This subject was alluded to when the last-named disease was under consideration. Westphal,[1] who was the first to give special attention to this matter, does not believe that there is any direct relation between the morbid process in the cord and that in the brain. Neither of them is, in his opinion, secondary to the other. They simply coexist as the expression of an excessive proclivity to disease of the nervous system, just as any other two diseases may be present, one in the brain and the other in the cord, without there being any direct interdependence between them. This is undoubtedly correct. Locomotor ataxia is by no means uncommon in patients affected with the other forms of insanity. Several such cases have come under my own observation, and Dr. Patrick Nicol,[2] in an excellent memoir, has adduced several instances which have occurred in his experience.

As we have seen, the lesions in sclerosis of the posterior root-zones are not always confined to the original seat. Among other parts of the cord liable to be involved is the anterior tract of gray matter. Hence we have the more complete development of paralysis and the supervention of atrophy in the affected muscles. A remarkable instance of locomotor ataxia combined with muscular atrophy formed the subject of a clinical lecture,[3] which I delivered at the Bellevue Hospital Medical College, in the winter of 1871–'72. In this case there were electric-like pains, incoördination, ocular troubles, ptosis, double vision, plantar anæsthesia, etc. After about two years muscular atrophy set in, beginning in the left leg, then involving the right corresponding member, then the left arm, and finally the right upper extremity.

In this case the lesion of the posterior root-zones was the primary lesion, the anterior tract of gray matter subsequently becoming symmetrically implicated. There were no contractions like those present when the lateral columns of the cord are the seat of disease.

In the case of the woman Moli, reported by Pierret,[4] to which reference has already been made, there were also the combination of the symptoms due to the lesion of the posterior root-zones, and those resulting from the extension of the morbid process to the anterior horns of gray matter—the right side being the seat of profound muscular atrophy. On post-mortem examination it was found that the right anterior horn of gray matter in the dorsal and cervical regions was the seat of degenerative changes in the nerve-cells, many of which had disappeared. The horn was markedly diminished in size. These changes are shown in the accompanying figure (Fig. 86) from Pierret—*a*, the posterior roots; *b*, the internal radicles, the sclerosis being limited to their area; *c*, the right anterior horn of gray matter atrophied. This association of muscular atrophy with sclerosis of the posterior root-zones is to be explained by the fact, first pointed out by Kölliker,[1] that some of the internal fibres of the posterior roots pass toward the anterior horns of gray matter, and can be traced as far as the large cells forming the external group. The connection of the fibres of the posterior roots with the anterior horns of gray matter is also referred to by Lockhart Clarke[2] and Gerlach.[3]

---

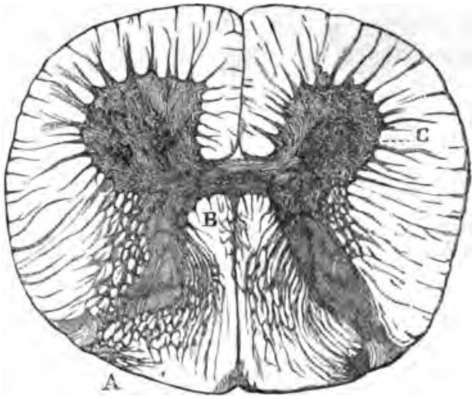
[1] "Ueber den gegenwärtigen Standpunct der Kentnisse von der allgemeinen progressiven Paralyse der Irren," Griesinger's *Archiv für Psychiatrie und Nervenkrankheiten*, Heft i., Band i., 1867.

[2] "On Progressive Locomotor Ataxy and some other Forms of Locomotor Deficiency, as found in the Insane," "West Riding Lunatic Asylum Medical Reports," vol. i., 1871, p. 178.

[3] "Clinical Lectures on Diseases of the Nervous System," New York, 1874, p. 156.

[4] "Sur les altérations de la substance grise de la moëlle épinière dans l'ataxie locomotrice considérées dans leurs rapports avec l'atrophie musculaire," *Archives de physiologie*, 1870, p. 590.

Fig. 86.



Treatment.—It must be remembered that locomotor ataxia often spontaneously remits in the violence of its symptoms. Indeed, the remission may at times amount to almost a complete intermission. But taking this fact into full consideration, I am quite sure that the disease is not in every case uninfluenced by medical treatment. A great many medicines have been recommended, and numbers of cures have been reported. Careful inquiry, however, suffices to show either that the alleged cures were merely instances of more or less complete remission, or that the cases were really not examples of the disease in question. To even mention the assumed remedies would be profitless labor.

---

[1] "A Manual of Human Histology," "Sydenham Society Translations," vol. i., 1853, p. 415.

[2] "Philosophical Transactions," 1853.

[3] Stricker's "Manual of Histology," American edition, New York, 1872, p. 645.

In the very earliest period of the disease ergot is calculated in some cases to be of decided benefit. It should be administered in doses of at least a drachm three or four times a day, and continued for several months. The bromide of potassium, sodium, or calcium, is an efficacious adjuvant. Under the combined use of these remedies I have repeatedly seen the electric-like pains diminish in violence or even altogether disappear. The gastric disturbances may often be alleviated by bismuth, or, what is usually still more efficacious, by the saccharated pepsin in doses of fifteen or twenty grains with each meal.

With these measures the primary galvanic current applied to the spine, on each side of the spinous processes, is an agent which ought to be used. Cases have been reported by Meyer, Benedict, and others, in which it alone has apparently effected cures—or arrest of the morbid process—and Rosenthal[1] speaks highly of its beneficial influence. I have used it with success in several cases in conjunction with the means previously mentioned. Ordinarily, it has not appeared to me to be of any material service.

The pains in the back and around the abdominal and thoracic regions may be combated with codeine in doses of from half a grain to one or even two grains, or with hypodermic injections of morphia.

If the case comes under observation when the motorial troubles are well marked, or if, after having used it for a month, no decidedly beneficial effect follows the treatment just specified, I omit the ergot, and frequently use instead, the nitrate of silver in doses of the quarter of a grain three times a day. According to Rosenthal,[2] Wunderlich, Charcot and Vulpian, Herschell, Klinger, Duguet and Vidal, have extolled its merits. This remedy has in my hands apparently proved serviceable in several cases which were well advanced, but I am not able to speak definitely on the subject, for the reason that with it bromide of potassium, and especially galvanism, were used. Two cases were cured by the combined remedies—one of them was that of a distinguished journalist, who, in the first place, was treated with ergot, and subsequently, when this medicine appeared to be of no further effect, with the nitrate of silver. At the present time, seven years having elapsed, this gentleman is well, free from pains, able to coördinate, and with no symptom of the affection remaining. The disease was first manifested by an epileptic paroxysm, and soon afterward ocular troubles made their appearance. The electric-like pains, abdominal constriction, and incoördination in the upper and lower extremities, were well marked. He was under treatment for about four months. The other case was that of a lady of this city. The disease in her began with pain in the back, and electric pains in the lower extremities. Ptosis, dilatation of the right pupil, and diplopia followed, and then gradual loss of sensibility in the soles of the feet, and difficulty in coördinating the muscles of the legs. The disease had lasted two years and a half when the patient came under my charge. She was treated with the nitrate of silver and the other remedies mentioned, for nearly a year, and throughout the whole period gradually improved till her recovery was complete. The nitrate of silver was suspended for a week after each month of its administration.

---

[1] "Klinik der Nervenkrankheiten," Stuttgart, 1875, p. 394.

[2] *Op. cit.*, p. 390.

In a third case ergot and nitrate of silver were given together without the bromide of potassium. This case was that of a gentleman, a merchant of this city, residing in Bridgeport, Connecticut. He had had ocular troubles, and was suffering from pains, incoördination, plantar anæsthesia, paralysis of the bladder, and swelling of the right knee, when he came under my charge, being sent to me by my friend Dr. Hubbard, of Bridgeport. The disease had then lasted only a few months. With the medicines, the constant galvanic current to the spine and spinal nerves was employed. He was entirely cured in less than three months.

In all cases inquiry should be made with reference to the existence of a syphilitic taint. If affirmative results follow the investigation, the iodide of potassium should be administered in gradually-increasing doses as recommended for acute spinal meningitis, or in combination with corrosive sublimate, according to the formula given on page 308, recollecting that galvanism is likewise to be used, and such other treatment as the special symptoms may seem to require. Two cases were cured by this treatment; one of them was that of a gentleman from the West —a fully-developed case—who had been treated by my friend Dr. Bumstead, for other syphilitic troubles, and who sent him to me for his spinal disease. The incoördination, plantar anæsthesia, pain in the lumbar region, and the electric pains, were all present, together with slight diplopia. He was under treatment for about ten months. I met him a few weeks since in a railway-car, the picture of health, and, as he told me, perfectly well.

The other case occurred in the person of a gentleman of this city, and was similar in general features to the preceding. A cure was obtained, after like medication, in six months.

In another case, after ergot had been used for several months without apparent benefit, the nitrate of silver was administered with the effect, to all appearance, of checking the further progress of the disease, and producing decided amelioration of the existing symptoms. The patient, a distinguished member of the dramatic profession, by my advice, withdrew from the stage, and being in Philadelphia, he consulted at my suggestion Dr. Weir Mitchell, who unhesitatingly confirmed my diagnosis. He took the nitrate persistently for about six months, and was so greatly improved that I gave my consent to his resuming his profession. There are now no pains; his coördination is good, and his general health leaves nothing to be desired.

In several cases I have obtained ameliorations by the use of phosphoric acid, phosphorus, and chloride of barium, but after extensive experience with these agents, I am unable to report any permanently good results.

If the vesical sphincter be paralyzed, belladonna may be used with advantage, preferably in the form of hypodermic injections of atropia gradually increased daily, from the one hundred and twentieth of a grain to the thirtieth.

Hydro-therapeutics in all forms, and faradization, have never, according to my experience, been of the slightest benefit, except as regards the use of the latter to the affected muscles. The ether-spray recommended by Jaccoud has been entirely inefficacious in my hands, and the same may be said of all plasters and embrocations.

One auxiliary means of treatment I have lately employed with advantage, and that is, keeping the patient as much as possible from using the groups of muscles which have lost their coördinating power, and requiring him, when he walks, to employ crutches to assist him. By systematically carrying out this plan the nervous force of the patient is not wasted, and a diseased organ, such as is his spinal cord, is not overtasked.

Lately I have employed, and thus far with apparently good results, the actual cautery to the spinal column. I have used it in eleven cases. The effect has been to lessen, and in three cases entirely to abolish, the electric pains and the feeling of constriction around the body. In one fully-developed case which I had before the medical class of the University of New York, the pains, which were of great intensity, ceased within a few hours after the first cauterization. Ten days subsequently I repeated the operation, the pains in the legs having returned, and again the relief was complete.

One other therapeutical measure seems to be of importance, and, if further experience sustains the views which we at present appear to be warranted in entertaining of its efficacy, it will be worth all other means combined. It is that of stretching the sciatic nerves. Langenbeck[1] was the first to do this for locomotor ataxia, and the results, not only in relieving the electric-like pains for which it was performed but in curing the ataxia, were such as to astonish the operator. Since then the operation has been performed by Esmarch with like favorable consequences. Certainly nothing in the whole range of neurotherapy is so contrary to our preconceived opinions, as these experiences and additional operations will be required before the procedure can be regarded as entitled to any serious consideration.

---

[1] "Berliner klinische Wochenschrift," No. 48, 1879.

VIII.

INFLAMMATION—SCLEROSIS—OF THE COLUMNS OF GOLL.

We have seen that the columns of Goll or posterior median fasciculi are generally the seat of a lesion simultaneously with, or more probably secondarily to, that which, existing in the posterior root-zones, causes the group of symptoms we call locomotor ataxia.

There is, however, no doubt that they may be the seat of primary disease, and, though the data are not yet sufficient to enable us to give the clinical history of the affection as fully as is desirable, we are not altogether without information on the subject. Our definite knowledge rests upon one case reported in full by Pierret,[1] and which I quote, greatly condensed as follows:

Catherine Magnaigat, when thirty years of age (1855), experienced numbness, "pins and needles," sensations of heat, and deep-seated pains in the extremities, especially the upper. There were also pains in the loins, obstinate headache, and a sense of tightness around the chest.

In 1860, vertigo and weakness of the lower extremities supervened. She did not distinctly feel the ground with her feet, and she was obliged to walk with a cane.

In 1863 she entered the Salpêtrière, and came under M. Charcot's care. Her condition was then as follows:

Tactile sensibility was diminished in the soles of the feet, the left especially. She could not walk without a crutch, which she used under her right arm. When she wished to go forward she experienced an impulse to spring or leap, and finally she advanced by a series of short steps, and felt as if impelled by a force she could not resist. When she closed her eyes while standing alone she maintained the erect position for a while, but would eventually have fallen unless supported. She was easily fatigued, and walking caused pains which compelled her soon to stop. Her feet seemed to stick to the ground when she made voluntary efforts to lift them. Sometimes, when she attempted to advance, she felt herself irresistibly drawn toward the left side. When after having taken a few steps she wished to go back, she turned round suddenly as if moved by a spring.

In 1866 she, for the first time, experienced constricting pains around the body low down, and electric-like in character. Soon afterward she felt similar pains in the anterior part of the thighs. Cutaneous sensibility was then diminished in the lower extremities.

---

[1] "Notes sur un cas de sclérose primitive du faisceau médian des cordons postérieurs," *Archives de physiologie*, 1873, p. 74.

The idea of the exact position of the limbs was not in the least impaired, and there was no incoördination. Such was her condition when in 1871 she died of pneumonia.

The post-mortem examination showed that the columns of Goll were throughout their whole extent in a state of sclerosis. It was most manifest in the dorsal region, where it, to a slight extent, invaded the posterior root-zones, to which circumstance, doubtless, the electric-like pains experienced by the patient were due.

The case would appear to show that sclerosis of the columns of Goll gives rise to certain symptoms in the lower extremities, however much the superior may retain their normal condition. In some cases of locomotor ataxia there has been noticed an unusual feeling of heaviness in the lower extremities, or a marked tendency to go backward, or a great feeling of fatigue after slight exertion, a marked incertitude in standing erect, or even an irresistible feeling of propulsion. In such instances, therefore, the columns of Goll were affected at the same time with the posterior root-zones. M. Pierret holds the opinion that these columns, to some extent, preside over motion.

The accompanying woodcuts (Figs. 87, 88, 89, 90) represent sclerosis limited to the columns of Goll, and are taken from M. Pierret's memoir. Fig. 87 refers to the cervical region, Fig. 88 to the dorsal. Fig. 89 shows the appearance of a section made at the level of the second dorsal vertebra, and Fig. 90 one taken from the upper part of the lumbar enlargement. The sclerosed portion is represented at *a* in each figure.

Fig. 87.

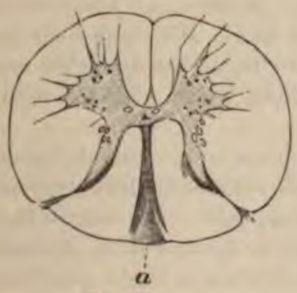


Fig. 88.

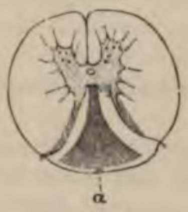


Fig. 89.

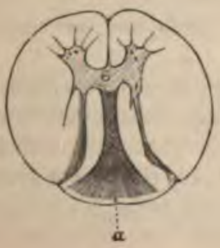
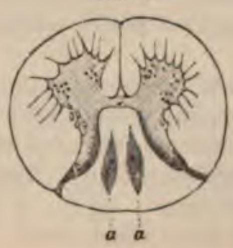


Fig. 90.



In the present state of our knowledge, all that we can do is to await further developments relative to the interesting points raised by the case which M. Pierret has so well studied.

IX.

DISSEMINATED INFLAMMATION OF THE SPINAL CORD—MULTIPLE SPINAL SCLEROSIS—SCLEROSIS IN PLATES—INSULAR SCLEROSIS.

Thus far we have considered the inflammatory affections of the spinal cord as they appear in one or another of the anatomical divisions which make up that nerve-centre. But we have now to engage ourselves with a lesion which has no fixed habitation, which is met with in the gray and white matter indiscriminately, and which occurs in distinct *foci*, patches, plates, or islets, in various parts at the same time or consecutively. This is what is known as multiple spinal sclerosis or sclerosis in disseminated plates—the *sclérose en plaques disséminées* of Charcot.

Symptoms.—Multiple spinal sclerosis generally first manifests its presence by more or less weakness in one or the other lower extremity. Before long the corresponding limb becomes involved; and, eventually, if the disorder continues to form additional centres of morbid action, the upper extremities are successively attacked.

At other times the first symptoms are connected with sensibility, and consist of the various sensations of numbness, tingling, "pins and needles," formication, and the like. Or these phenomena may make their appearance simultaneously with the paresis. The gait of a person affected with multiple spinal sclerosis is uncertain and titubating—like that of an individual slightly intoxicated. Although there is defective coördination, the patient stands as well with the eyes shut as open, and has no additional difficulty in walking in the dark or with the eyes closed.

The paralysis advances, but there are no marked disturbances of sensibility, and the numbness which may have been present to some extent in the early stage usually disappears. The patient is, therefore, sensitive to changes of temperature, to pain, and to pressure. Pains are very uncommon. Occasionally, there are slight painful sensations in the paralyzed parts, but they are temporary.

The general health usually remains good, and the mind is unaffected.

Later, in the course of the disease, rigidity or contraction makes its appearance in the paralyzed limbs, or both these conditions may coexist in the same extremity, some of the joints being contracted, and others rigidly extended. The tendency is for these conditions to become permanent. Again, there are violent tonic convulsions in the paralyzed limbs which may be spontaneous, but which are readily excited by impressions made upon the skin of the affected extremities, or even sometimes by mental emotions. They may precede, or coexist with, or follow the permanent contractions.

In some instances these phenomena are not met with. They were absent in the case of Dr. Pennock, reported by Drs. Morris and Mitchell; in a case under my own charge, and in which I made an examination of the cord soon after death; and in a case reported by Friedreich,[1] in which multiple spinal sclerosis existed in conjunction with the lesions of locomotor ataxia.

When present, as they generally are, these permanant contractions of the muscles exhibit different phases in the upper and lower extremities. In the former the flexors predominate over the extensors, while in the latter the extensors prevail. The spasmodic tonic convulsive movements of the limbs are especially met with in the lower extremities, the upper being rarely their seat.

After a time, which may vary from three or four to fifteen or twenty or even more years, the limbs become almost entirely paralyzed, and the contraction and rigidity are still more strongly marked. Whatever voluntary movements the patient is capable of executing now cause pains in the parts. The sensibility usually, however, even at this period remains but little affected. Reflex excitability generally exists though perhaps slightly impaired; sometimes it is altogether lost. The bladder and the sphincter ani retain their power to the last. Bed-sores form on the parts subjected to pressure as the patient lies in bed, and death eventually ensues, either from exhaustion or from some intercurrent affection.

Such is a description of multiple spinal sclerosis as it is ordinarily encountered—and it must be confessed that the clinical features are not very striking or peculiar. But even this type, imperfect as it is, is subject to great diversities. Sometimes there are violent pains of an electric-like character simulating those which are so prominent a feature of locomotor ataxia and like them resulting from the implication of the posterior root-zones in the lesion. Sometimes the superior extremities are attacked first. Again, anæsthesia constitutes a prominent feature, and the phenomena ordinarily present may be more or less modified in extent and intensity in different cases.

In their very excellent monograph on the subject, MM. Bourneville and Guérard,[2] in detailing the symptomatology of the spinal form of disseminated sclerosis, say:

"After a variable time the superior and inferior extremities become the seat of rhythmical agitations, which are only present, however, when spontaneous or voluntary movements are made. In the state of repose the members are not affected with any tremor."

---

[1] "Ueber degenerative Atrophie der spinalen Hinterstränge," *Archiv für pathologische Anatomie und Physiologie*, 1863, p. 433.

[2] "De la sclérose en plaques disséminées," Paris, 1869. p. 61.

In this connection I desire to repeat what I wrote five years ago,[1] that "tremor is never observed in spinal sclerosis of any form, diffused, multiple, or cortical, unless the pons Varolii or superior ganglia of the brain are implicated. In the only case of this latter form published—that of Vulpian [2]—the sclerosis extended throughout the whole length of the cord, and likewise involved the pons Varolii, cerebellar peduncles, and other intra-cranial organs, besides being accompanied with well-marked spinal meningitis. The tremor observed at a late period of the disease cannot, therefore, be ascribed to the lesion of the cord below the medulla oblongata."

Of the cases cited by Bourneville and Guérard in which post-mortem examinations were made, one from Vulpian and one from Morris and Mitchell, in which the lesions were restricted to the cord, there was no tremor at any time in the course of the disease; and in a case of my own already cited, and which will be still more specifically referred to hereafter, in which the cord was the seat of several islets of sclerosed tissue, tremor had never been a feature of the symptomatology.

As we shall see hereafter, when we come to the consideration of the cerebro-spinal form of the disease—multiple cerebro-spinal sclerosis—tremor constitutes one of the most prominent phenomena of the affection. We have already seen that it is a marked symptom of the purely cerebral type of the affection. I am quite sure, however, that in the disease we are now considering, restricted as its lesions are to the spinal cord, rhythmical tremor is not encountered.

Causes.—The causes of multiple spinal sclerosis are not well understood. In a case fully reported by Vulpian,[3] the affection appeared to have been induced by a sprain of the left ankle. The extremity remained weak, and three years afterward the patient had a fall, and then the right lower extremity became weak and subsequently the right upper extremity. The left upper extremity was not affected for several years.

In the case of Dr. Pennock, reported by Drs. Morris and Mitchell, the disease began while the patient was busily engaged in professional studies.

In the case in which I verified the existence of the disease by post-mortem examination, it was apparently caused by exposure to cold and dampness.

It is probable that blows on the spine, concussions—such as are produced by railway accidents—and the gouty and syphilitic diatheses—

---

[1] First and subsequent editions of this work, p. 473.

[2] *Op. cit.*, p. 64, *et seq.*

[3] "Note sur la sclérose en plaques de la moëlle épinière," *Union médicale*, 1866, Juin 7, 9, 14, et 19, obs. i.

may induce multiple spinal sclerosis. There is in reality no reason, to my mind, why all the influences which are capable of causing the diffused forms of sclerosis which have been considered, may not also cause the disseminated variety. But it is difficult to arrive at any definite information relative to this matter, so long as the clinical features of the disease are so little characteristic.

Diagnosis.—There is very little in multiple spinal sclerosis sufficiently pathognomonic to aid us in our diagnosis of the affection. The symptoms in some cases are identical with those of symmetrical lateral sclerosis, in others they resemble those of locomotor ataxia, as in the two cases reported by Friedreich, to one of which allusion has already been made. In the present state of our knowledge, therefore, I am afraid we must wait for the scalpel and the microscope to determine with any degree of accuracy the diagnosis of multiple spinal sclerosis.

Prognosis.—The disease is not one which is directly calculated to cause death. All the patients known to have died while subject to it, succumbed to some intercurrent affection, such as bronchitis, dysentery, typhoid fever, and pneumonia. It undoubtedly tends to weaken the vital powers, and hence is indirectly the cause of a fatal result. So far as any prospect of arresting, by therapeutic means, the tendency to the formation of other islets of inflammation and sclerosis, or of restoring the integrity of the cord is concerned, there does not appear to be much hope. For, though its progress is in many cases slow, and in others seems, at times, to be self-limited, it pursues its course unamenable, so far as we know, to medical treatment. In the diffused forms of spinal sclerosis there is but one centre of morbid action; in the disseminated there are several, which, if not coexistent, tend, through an inherent proclivity, to be produced indefinitely. To this circumstance is due the fact that the prognosis of the disease under consideration is more unfavorable than that of sclerosis of the posterior root-zones or even symmetrical lateral sclerosis.

Morbid Anatomy and Pathology.—Multiple spinal sclerosis consists in the dissemination through the cord of masses of sclerosed tissue, which have resulted from the proliferation of the neuroglia and the consequent atrophy and disappearance of the proper nerve-elements. They are of a gray color, of increased consistence, of irregular size and form, and may exist in any part of either the gray or white tissue of the cord; often, however, manifesting a tendency to involve the two lateral halves of the cord symmetrically.

In the case reported by Vulpian, the volume of the cord was evidently diminished, and on different points of its surface exhibited an ashy-gray coloration. The antero-posterior diameter of the cord was markedly lessened at those places where the islets of sclerosed tissue existed.

In this case there had been progressive paresis, rigidity, and contraction, with extension of all four limbs, without tremor of any kind. The alterations were found in the anterior, lateral, and posterior columns, and in the anterior and posterior horns of gray matter.

In the case of Dr. Pennock, reported by Drs. Morris and S. Weir Mitchell,[1] the sclerosed tissue was confined mainly to the lateral columns. The posterior were involved to a very small extent. In this case there were partial anæsthesia, gradually-advancing paralysis implicating all four extremities, and paralysis of the bladder. The intellectual faculties were never affected in the least. The course of the disease was progressively onward, and, though there was toward the last a total loss of voluntary power below the neck, reflex action remained unaffected. There were no tremors with or without voluntary movements. In regard to this case, Dr. Mitchell, who made the microscopical examination, remarks that there were :

"1. Integrity of mental and moral manifestations.

"2. Absolute loss of voluntary motive power below the head, or rather below the neck.

"3. Sensation nearly perfect.

"4. Respiration good ; reflex motion preserved and exhibited in the form of spasm or irritation of certain parts of the skin."

All of which are what we should expect to find in sclerosis almost entirely confined to the lateral columns.

In the case which I have mentioned as coming under my own observation, the patient, J. H., consulted me in the winter of 1869-'70. He was then unable to walk without a cane and the assistance of an attendant. He had previously been treated at a water-cure establishment, and more recently by the Swedish movement-cure, and of course without benefit. The symptoms were mainly connected with motility. Both lower extremities were paralyzed ; the bladder was inactive, but not the sphincter, and there was obstinate constipation. There were occasional fibrillary contractions of the paralyzed muscles, and at times pain in the back and limbs—never, however, of any great degree of severity. There were no tremors, either with or without voluntary motions.

The patient obtained very little benefit from the treatment to which I subjected him, and I advised him to return to his home in Ohio. A few months afterward, he died.

The dorsal, lumbar, and sacral regions of the cord were sent to me for examination by his physicians, Drs. Ramsey and Bishop, of Delhi, Ohio. In a letter, the latter informed me that the vessels of the pia mater were injected.

The cord arrived in good condition, having been carefully preserved in strong alcohol. Upon inspection, the antero-lateral columns in the middle and lower dorsal regions to the extent of three and a half inches were seen to be of a grayish tint, which became deeper in shade from above downward. Below this, at the junction of the dorsal with the lumbar portion, was another patch two and a half inches in length, and also involving the whole superficies of the antero-lateral columns; and, separated from this by a portion of apparently healthy tissue, was another discolored, irregular patch, an inch and a half in length, along the left antero-lateral column; and, below this, a similar tract, two inches and an eighth long, involving the right antero-lateral column. The difference in consistence between these patches and the other parts of the cord was very decided, and the white striæ were well marked. The sacral portion of the cord presented no abnormal appearance to the naked eye.

---

[1] *American Journal of the Medical Sciences*, July, 1868.

Sections of the cord were then made through the sclerosed portions; and it was seen that the gray matter was only involved where the horns approached the surface; and that, wherever a lesion existed, the normal contour of the sections was altered so as to make them sub-ovoidal, and thus to lessen the circumference. The greatest depth of any part of a sclerosed region was two-twelfths of an inch, and this was in the superior patch. The average thickness was about the one-twelfth of an inch.

The whole cord in my possession was then immersed in a solution of chromic acid in water, and left there for a month to harden. Immediately previous to examining with the microscope, the sections were colored by an ammoniacal solution of carmine. Under a twelfth-inch objective, it was seen that, throughout the whole extent of the sclerosed portion of any section, the nerve-tubes had entirely disappeared; and, wherever the gray substance was affected, the nerve-cells were diminished in number. In the place of these elements were connective tissue, a large quantity of molecules, and connective-tissue cells in great abundance.

In several sections taken from the dorsal, lumbar, and sacral regions, and which were apparently normal when viewed with the naked eye, the neuroglia was found to be in excess, and the nerve-tubes in a state of disintegration.

The gray matter, except in those sections made through the part where the sclerosed portion extended from the white matter to it, was uniformly healthy, and in no part were the posterior columns involved.

In this case there was no tremor, although it was clearly one of multiple sclerosis, probably entirely confined to the spinal cord. At no time had there been head-symptoms of any kind. Histologically, therefore, we see that the sclerosed tissue consists mainly of an excessive amount of connective tissue—the neuroglia of Virchow. The cells are increased in size, and the nuclei are larger and much more numerous than in the normal condition. The capillaries are thickened, from the deposition on their walls of several layers of rounded cells.

The effect of this morbid process is to compress the nervous filaments and to cause their atrophy. The fluid portion undergoes fatty degeneration, and the axis cylinders become disintegrated. Still, however, they present somewhat of their characteristic color and consistency, and appear as white striæ traversing the morbid tissue.

The membranes often exhibit evidences of inflammation, and are thickened, opaque in spots, or red in some cases, while in others they are adherent to each other and to the cord.

Treatment.—Something can be done to mitigate the violence of the symptoms. Hypodermic injections of atropia have often a happy effect in diminishing the force and frequency of the tonic contractions. The nitrate of silver has been used by M. Piorry with temporary good results.

The primary or galvanic current has, in my hands, been of like efficacy in lessening the contractions or spasmodic rigidity, but with this agent, as well as with the others mentioned, there can be no great certainty that we are dealing with a case of multiple spinal sclerosis. We are, therefore, forced to treat symptoms instead of lesions.

Still, for the cure of the disease we may attempt the measures recommended for symmetrical lateral sclerosis, but with even less prospect of success. I should be disposed to use, with thoroughness and persistency, the actual cautery in the manner recommended when discussing the treatment of locomotor ataxia.

X.

SECONDARY INFLAMMATION AND DEGENERATION OF THE SPINAL CORD.

It is a well-recognized fact that disuse of an organ promotes its atrophy and degeneration. A muscle, which from any cause is rendered incapable of contracting, becomes smaller, and its fibrillæ undergo conversion into fat. The same law applies to other organs, and among them the spinal cord. Whatever interrupts the passage of the normal excitations through its columns causes degeneration. Thus, if there be a cerebral hæmorrhage, preventing the action of the brain on the muscles, the anterior columns of the cord, not being stimulated by their accustomed excitation, undergo the change mentioned. If the cord itself be the seat of a lesion, or the posterior nerve-roots, and perhaps even the nerves or muscles, the posterior columns above, no longer being required to convey impressions to the brain, suffer atrophy and degeneration. To this alteration, which is not itself a primary disease, but which is always, in its very nature, consecutive to lesions in superior or inferior parts of the nervous system, the term secondary degeneration has been applied.

The fact that the spinal cord is affected by lesions of the brain was observed by Cruveilhier,[1] who, however, failed to notice any consecutive change in the cord below the decussation of the pyramids.

L. Türck[2] was the first specially to inquire into this important subject, and, in a series of memoirs extending through the years from 1851 to 1855, he showed that the cord underwent secondary degeneration, both from lesions of the brain and of its own substance. Since these memoirs, other pathologists, among whom MM. Charcot, Turner, Rokitansky, Vulpian, Cornil, and Lancereaux, may be mentioned, reported cases, but no one has investigated the subject with so much thoroughness as M. Bouchard.[3]

Symptoms.—The most important symptoms referable to secondary degeneration of the cord from cerebral lesions are muscular contractions. These are not the contractions which sometimes exist from the very inception of a hæmorrhage, for instance, but those which come on at a later period of the disease, and which, like the first, have generally been thought the consequence of irritation existing about the cicatrix. Bouchard, however, shows very clearly that they are the result of secondary changes taking place in the spinal cord, and the clinical history of which has not hitherto been carefully studied. They are very frequent. Of thirty-two cases of old hemiplegia analyzed by Bouchard, they were present in all but one. From my own experience I think it is safe to say that it is very rare to meet with a case of hemiplegia of over a year's duration in which they do not exist.

In examining a patient suffering from an old hemiplegia, it is common to find the forearm of the paralyzed side flexed on the arm. Frequently, also, the fingers are bent into the palm of the hand, the hand flexed on the forearm, and the whole member carried across the front of the body, and held firmly against it by the contraction of the pectoralis major muscle. In such a case we find the muscles atrophied, hard, and stretched to an extreme degree of tension. Rectification of the position is, to a great extent, impossible by the voluntary efforts of the patient. He may be able to accomplish a little motion, and to do still more by using the sound hand to extend the affected arm; but, if the hemiplegia has been of considerable duration, the range of his motility, with or without assistance, is very small, and is sometimes nothing. I have found that the electric contractility of such muscles is diminished in some of their fibres, unaffected in others, and exalted in others, so that, when the electrical stimulus is applied, a hard, irregular, and knotty contraction is obtained.

---

[1] "Anatomie Pathologique," liv. xxxii., p. 15.

[2] "Ueber secondäre Erkrankung einzelner Rückenmarksstränge und ihrer Forsetzungen zum Gehirne," "Sitzungsberichte der Kaiserlichen Wiener Academie," 1851.

[3] "Des dégenérations secondaires de la moëlle épinière," *Archives générales de méd.*, 1866.

This condition is much more common in the muscles of the upper extremity than in those of any other part of the body. The muscles of the trunk are never involved, and, unless, as Bouchard appears to think, the muscles of the face are occasionally affected, the alteration is entirely confined to the extremities. Of these, the upper are much more frequently its seat. Thus, of the thirty-one cases of rigidity with contraction, studied by Bouchard, the upper extremity was implicated in all, and the lower but in fourteen. In none of his cases was the lower extremity affected without the upper also participating, and he lays this down as an invariable occurrence. I, however, had a patient under my charge, a gentleman from the West, who five years previously had had an attack of cerebral hemorrhage which rendered him hemiplegic on the left side. There was not the slightest contraction of the muscles of the left upper extremity, but the toes of the left foot were strongly flexed, and the sole of the foot turned inward by the contraction of the flexor longus digitorum, and the tibialis posticus.

In another case, the only contraction was in the flexors of the toes of the left foot. So great a degree of inconvenience did this condition induce, rendering as it did walking or standing extremely painful, that, after ineffectual efforts at extension, I amputated the second toe, to the great relief of the patient.

In another instance under my charge, the upper extremity was not carried across the front of the body, but was drawn backward by the contraction of the latissimus dorsi.

The period at which these secondary contractions begin in cases of hemiplegia has been carefully studied by Charcot, and he has ascertained that they habitually make their appearance during the second month. The fingers are usually the first to be affected from the contraction of the flexor muscles in the forearm. A symptom mentioned by Bouchard, as sometimes occurring, a trembling in the arm when it is raised, I have witnessed many times. Sometimes, especially, in the lower extremity, it is only necessary for the patient, while the foot rests on the ground, to give the leg a few voluntary movements, when the agitation is kept up for a long time involuntarily.

Atrophy of the paralyzed muscles may be one of the secondary results of brain-disease ; as we have seen, it is of a primary spinal affection.

When the cord itself is the seat of primary disease, the lateral columns below undergo degeneration, and the muscles become permanently contracted. Many cases of distortion which ensue on sclerosis, tumors, and other lesions, are the result of this secondary degeneration. M. Charcot is of the opinion that the epileptiform attacks sometimes met with in hemiplegics may result from these secondary descending degenerations affecting the peduncles, the pons, and the medulla oblongata.

No symptoms referable to ascending secondary degenerations—those of the posterior columns—have been recognized.

Causes.—Secondary degeneration of the spinal cord may result from primary lesions of the cerebral hemispheres, of the cerebral peduncles, of the pons Varolii, of the medulla oblongata, of the spinal cord itself, and of the posterior roots of the spinal nerves. The immediate causes are the loss of the due supply of arterial blood, and the arrest of nutritive action from deficient nervous influence.

The **Diagnosis** calls for no special consideration.

Prognosis.—This is not so unfavorable as might at first sight be supposed. Bouchard concludes that a cure is possible even in severe cases. In five cases which came under his observation, and in which there was complete paraplegia due to the compression of the cord in Pott's disease, complete cures were obtained in four, and a partial cure in the other. In the four entirely successful cases as regards the restoration of sensibility, and the power of motion to the paralyzed limbs, there were contractions. He therefore concludes that the nerve-fibres of the cord, like those of the peripheral nerves, may be regenerated.

My own experience is to the same effect. In cases of muscular contractions resulting from cerebral hemorrhage, and secondary degeneration of the cord, and in like troubles due to primary lesion of the cord itself, followed by secondary degenerations, I have several times succeeded in effecting the complete relaxation of the contracted muscles, and the entire restoration of sensibility and the power of motion to the paralyzed limbs.

Morbid Anatomy and Pathology.—Secondary degeneration is generally found in the white substance, the gray being seldom affected. This might certainly have been expected, owing to the fact that it is the conducting power of the cord only that is lessened, and, as this power resides almost entirely in the fasciculi of the white substance in the antero-lateral and posterior columns, it is here that we ordinarily find the lesions. When a fibre belonging to the white substance is injured, either in the cord or in its intra-cranial prolongations, the secondary degeneration ensues either above or below the seat of the primary lesion, but it extends through the entire length of this portion to its central or peripheral extremity, according as it involves sensory or motor filaments. To these two varieties, the terms ascending and descending degeneration are applied. The affected fibres alone are changed, and the alteration extends throughout their whole length. But, as the white fibres are constantly receiving other fibres which have had no initial injury, the secondary degeneration becomes relatively less the greater the distance is from the seat of the primary lesion.

The morbid condition depends upon three processes: atheroma of the capillaries and the formation of granular corpuscles in the degenerated tissue; the degeneration and atrophy of a greater or less number of nervous filaments; the proliferation of connective tissue which takes the place of the nerve-tubes. These changes are similar to those which occur in the several forms of sclerosis, to which attention has already been directed, and are essentially inflammatory in character.

When there is atrophy of the paralyzed and contracted muscles as a result of secondary degeneration of the cord we may be very sure that the anterior horns of gray matter are involved. Charcot[1] cites a case which he reported to the *Société de Biologie*, in which a woman aged seventy was suddenly struck with left hemiplegia, occasioned, as the post-mortem examination showed, by a cerebral hæmorrhage seated in the centrum ovale of the right hemisphere. Contraction of the paralysed muscles supervened very soon, and, two months after the attack, the muscles of the inferior as well as of the superior extremity began to atrophy at the same time that their electric contractility was notably diminished. The muscular atrophy advanced with great rapidity, and simultaneously the skin on the paralyzed parts, when submitted to pressure, was the seat of numerous bullæ and even erosions.

The examination of the spinal cord revealed the existence of a descending sclerosis, occupying the left side, and presenting its ordinary features. But in addition, at several points of the cervical and lumbar enlargements, the anterior horn of gray matter of the same side exhibited evidences of an inflammatory process, and at these points the large nerve-cells had undergone a marked degree of atrophy.

Similar cases have been reported by Hallopeau.

Treatment.—The best results in my experience have been obtained from the use of the primary galvanic current to the cord, the same or the induced current to the muscles, forcible extension and flexion of the contracted limbs, and the internal administration of nitrate of silver, chloride of barium, and cod-liver oil. It will generally be found that the opposing muscles are more or less paralyzed, and that great good may be effected by stimulating them with the primary or induced currents. The division of tendons is never necessary, unless for the rectification of distortions of the toes or fingers. Sometimes the toes are strongly flexed against the sole of the foot, rendering it almost impossible to walk, from the pain produced by the dorsal surface being brought in contact with the ground, and hence obliged to bear the weight of the body. In such cases the tendons may with propriety be divided, unless the toes can be kept extended by some convenient prothetic apparatus, or as in the case under my care, to which reference has been made, the toe may, if necessary, be amputated.

Passive exercise of the affected muscles will do much to restore them. Indeed, I am disposed to think that in some cases the tendency to contractions, which is so evident in most cases of cerebral hæmorrhage, may be obviated by passive movements of the paralyzed limbs being begun at an early period, and continued for a long time.

---

[1] "Leçons sur les maladies du système nerveux," 1874, p. 245.

CHAPTER VI.

NON-INFLAMMATORY SOFTENING OF THE SPINAL CORD.

SOFTENING of the spinal cord is, as we have seen, the common termination of acute myelitis, in which connection it has been sufficiently considered; but it may originate primarily, and in that event possesses a clinical history very distinct from that of acute inflammatory softening.

Symptoms.—The first symptom usually noticed in softening of the spinal cord is numbness in those parts of the body below the seat of the lesion. Soon after the occurrence of this symptom there is weakness of the same parts, and then the deficiency of sensation and the feebleness of motor power advance together, both gradually becoming more and more strongly marked. There are no muscular twitchings, no contractions of the limbs, no pains either at the seat of the disease or in the paralyzed limbs.

The bladder very soon becomes involved, and the patient finds that, when he attempts to urinate, the stream is not so strong as it once was, and that he is obliged at times to use the expulsive force of the abdominal muscles in order to complete the evacuation of the bladder. Gradually the contractile power of this viscus becomes less, and finally is altogether lost.

The sphincter generally participates. The desire to urinate becomes more frequent, and when the inclination is felt the patient must at once yield to it. Eventually the bladder likewise becomes entirely paralyzed, and then there is neither the ability to expel the urine nor to retain it, and consequently it dribbles away constantly.

Sometimes the first evidence of softening of the cord is perceived either in the bladder or its sphincter, and it may be restricted to these parts for a considerable period. I have a patient at the present time under treatment for what I have no doubt is softening of the cord, and in whom the bladder-troubles were the only notable symptoms for over two years.

The intestines are similarly affected, and the bowels are either obstinately constipated or the sphincter ani is relaxed, leading to fecal evacuations as soon as the contents reach the rectum.

Reflex excitability is weakened from the first, and gradually disappears, unless, as is rarely the case, the gray matter be unaffected.

The progressive advance of the disease reduces the patient to a condition of utter helplessness. He is unable to walk, sensation is abolished in the paralyzed limbs, his urine and fæces are passed involuntarily, bed-sores occur, the venereal appetite is extinct, or, if it should remain, erections are impossible, and the parts of the body below the seat of the disease are to all intents and purposes cut off from communication with the parts above. This condition may last for years without a fatal termination ensuing, but intercurrent affections, especially resulting from the bladder-troubles, may eventually cause death.

Such is the course of spinal softening when the lesion is low down and involves both antero-lateral and posterior columns. When it is higher up, the symptoms are also referable to the thoracic extremities, and to the muscles concerned in deglutition and respiration. There are likewise visceral disturbances.

When the lesion mainly affects or is confined to the antero-lateral columns, the symptoms manifested are in intimate relation with the known physiological functions of the region in question. Thus the power of motion in the limbs below the softened portion of the cord gradually becomes less, the gait is from the first staggering, and, though even at a late stage the patient may be able to move his limbs while lying down or sitting, he cannot support the weight of his body upon them. When he tries to stand without extraneous aid, it is seen that he is especially weak in the knees and ankles. There is no more difficulty in standing or walking with the eyes shut than when they are open.

This paralysis of motion, in which the bladder generally participates, may be of the most profound degree, and yet sensibility be perfect. A gentleman was under my care in whom I diagnosticated softening of the cord in that part extending on the right side from the second dorsal vertebra downward probably as far as the fourth sacral, while on the left side it began at about the fourth lumbar and extended downward probably as low as the fourth sacral. I gave the lesion these topographical limits for the reason that on the right side the muscles supplied by the crural and sciatic nerves had lost their electro-muscular contractility, while it certainly did not extend above the origin of the ilio-hypogastric nerve, as the lower part of the rectus abdominis, which receives its motor power through this nerve, retained its contractile power. On the left side the muscles supplied by the crural nerve were possessed of their normal motor power, while those supplied by the sciatic had lost their contractility. It was, therefore, very certain that on this side the lesion did not extend above the fourth lumbar, the lowest spinal nerve contributing to the formation of the crural.

I was able also to restrict the morbid process entirely to the antero-lateral columns, for in no part of the skin below the upper supposed limit of the lesion was there any loss of sensibility. The least impression made upon the skin was felt. Tickling the sole of the foot excited laughter, but no reflex movements. I was therefore able to determine that the gray matter was involved. The bladder was paralyzed, and its sphincter likewise. The sphincter ani was also deprived of its contractile power to a great extent.

The patient died at Cape May, and I had no opportunity of making a post-mortem examination. Probably, however, the lesion was essentially that which I have described. In all cases of spinal softening involving the antero-lateral columns, the electro-muscular contractility is soon lost, so that even the strongest induced or primary currents fail to cause contractions.

As regards the implication of the posterior columns, there is an equal facility for determining the fact from a consideration of the symptoms. The functions of these columns are intimately connected with sensation, and when such a morbid process as softening is set up in them the symptoms are those which indicate impairment of the cutaneous and muscular sensibility. Thus, in a gentleman formerly under my charge, there had been going on for several months a morbid action in the spinal cord unattended by any prominent symptoms except anæsthesia. There had never been pain or any derangement of motility, but simply a gradually-increasing loss of sensibility in both lower extremities and in all the other parts of the body below the upper limit of the seat of the lesion.

He was unable to walk in the dark or with his eyes shut, or to stand alone with his eyes closed and his feet close together, for he obtained no idea of his position unless he could have the aid of his eyes or hands.

He had full power over the bladder and voluntary control over its sphincter and that of the rectum, but he never experienced the desire to urinate, did not feel the flow of urine through the urethra, nor the passage of the fæces through the anus, and evacuated his bladder and bowels at stated periods merely from the knowledge acquired by experience that it was time to do so.

Examination with the æsthesiometer showed that the upper limit of the lesion on both sides was in that part of the cord from which the second lumbar nerves are derived, for the loss of sensibility was apparent in all those parts supplied by the crural and sciatic nerves, both as regarded the skin and the muscles. Very weak faradaic currents caused muscular contractions, but the strongest which it was possible to obtain from a powerful machine produced no pain.

There was no muscular incoördination, neither had there ever been electric-like pains in any part of the body. The patient died in 1873. For a year previously he had exhibited indications of insanity, and finally committed suicide by hanging himself to his bedpost. A post-mortem examination was made of his brain, but the physician who then had charge of the case thought it too great a trouble to examine the cord, and thus an opportunity for studying what must necessarily have been important lesions was lost.

In this case there was, I think, ample reason to diagnosticate a lesion of the posterior columns without any implication of the antero-lateral. The reasons for believing this lesion to have been softening will be indicated under the head of diagnosis.

Causes.—The causes of spinal softening are not very clearly understood. Doubtless it arises as a consequence of acute myelitis, but it is often an independent and apparently a primary affection, being unpreceded by any obvious symptoms indicative of spinal derangement. Such influences as give rise to cerebral softening will, in all probability, cause spinal softening, and among them must be placed obliteration of blood-vessels from embolism and thrombosis. The actual occurrence of occlusion of spinal vessels from either of these causes has not, however, so far as I am aware, been demonstrated. The further etiology of spinal softening is not as yet a matter of any certainty, though I think several cases that have been under my observation could reasonably have their cause laid to excessive sexual indulgence.

Diagnosis.—The diagnostic marks of most value in cases of supposed spinal softening are the absence of sensory and motor excitement. Thus there are no pains referable to the back or other parts of the body, no hyperæsthesia, no twitchings, no spasms, no contractions, no exalted reflex actions. And this is the case in that form of the disease involving the whole thickness of the cord, or in either of those limited to the anterior or posterior columns. There is no other affection of the spinal cord which is not characterized, at some time or other of its progress, by irritation either of the sensory or motor nerves, or of both, excepting some cases of spinal anæmia giving rise to the categories of symptoms previously considered. The clinical history of such cases, and the comparatively light character of the phenomena, will serve to distinguish them from those in which the lesion is softening.

Prognosis.—The prognosis is always unfavorable as regards recovery and complete restoration, but spinal softening is not necessarily a fatal disease. At least, I have seen cases which had existed for many years, and which apparently had no elements of a fatal termination about them. But they were instances in which the seat of the disease was in the lower dorsal, or lumbar or sacral region of the cord. When it is higher up, the prospect of death ensuing is more probable. The restoration of the cord to its normal structure is impossible, and the patient lies paralyzed either in sensation or motion, or both, according to the situation and extent of the lesion, in a condition similar to that of a person who has received a wound inflicting irreparable injury on the cord. Such persons, as is well known, frequently live for many years afterward—then die of some entirely different disease. There is nothing about spinal softening calculated to produce exhaustion, unless it be the tendency which exists to cystitis from paralysis of the bladder, and the consequent inflammation liable to be set up from the action of the retained urine. Care, however, will very greatly diminish the danger from this source. I have had a number of patients under my charge who had not, for many years, had a passage of urine from the bladder which was not effected with the catheter, and they had, in all that time, suffered no marked inconvenience.

Morbid Anatomy and Pathology.—The appearance of a softened portion of the spinal cord to the naked eye has nothing very peculiar about it. When examined as to its consistence, it is seen to be sometimes as soft as cream, at others scarcely altered in the resistance which it offers to the touch. In the first instance, when the lesion involves the gray and white matter together, section does not show the peculiar double crescentic arrangement of the former tissue, but it appears to be blended homogeneously with the white substance which surrounds it.

Microscopically it is seen that the nervous tubules constituting the essential anatomical elements of the white substance are broken up, and no vestige of them remains in extreme cases—oil-globules and bodies called granule-masses, the constituent of which is fat, having taken their place. In the gray substance the nervous cells are destroyed, and oil and fat have made their appearance in large amount. Even the neuroglia or connective tissue of the cord exhibits a similar disintegration and regressive metamorphosis. These changes impair the functions of the cord, both as a nervous centre and as a structure serving for the transmission of sensory impressions to the brain, and of nervous force from it. When the disintegration is complete, the effect is the same as if the cord had been entirely divided by a cutting instrument.

Treatment.—There is nothing to be done which can by any possibility restore the integrity of the spinal cord after the process of softening has fairly entered upon its course. In the very early stages, if patients apply for treatment at these times, something may perhaps be accomplished by the use of phosphorus and strychnia, but the symptoms come on so insidiously and gradually that the subject of them rarely has his apprehensions excited till it is too late to do any thing toward arresting the disease. And even when we do see cases which in appearance exhibit the symptoms met with in spinal softening in its initial stage, and which recover under treatment, there must always be a doubt in regard to the accuracy of the diagnosis—for many cases of temporary anæsthesia and impairment of motility are due to anæmia of the cord, the result of the causes set forth in a previous chapter.

The patient, however, may be made comfortable to such an extent as to materially prolong his life. Care should to this end be taken that he does not sustain a fall or suffer an injury whereby the diffluent portion of the cord would be disturbed in its anatomical relations, and the danger of an attack of acute meningitis or of myelitis incurred. Bed-sores should be prevented, or, if they occur, treated according to the methods previously mentioned, and full instructions should be given in regard to emptying the bladder with the catheter at regular times, and of going to stool at the same hour every day. Locomotion may be provided for by some one of the chairs devised for the use of paraplegics. As there is little, in softening of the cord situated below the origin of the phrenic nerves, which is directly calculated to destroy life, there is no reason why, with the adoption of proper measures, the patient should not enjoy a measurable degree of comfort for many years. Probably the event most apt to occur is acute or chronic cystitis from paralysis of the bladder, but attention to the injunction above given will do much toward lessening the liability to this affection.

CHAPTER VII.

TUMORS OF THE SPINAL CORD.

FOLLOWING the example of Jaccoud, I shall consider under one head, tumors of the cord, of the membranes, and those which, growing from the interior surfaces of the vertebræ, may compress the cord, and thus interfere with its functions by deranging its structure. In the present state of our knowledge, we have not many exact data by which to discriminate between these several growths.

Symptoms.—The phenomena which result from intra-spinal tumors, like those due to congestion, are of two categories, resulting as they do either from irritation or compression. Under the first head are embraced pain in the back, in the limbs, and in the viscera, if the posterior columns are mainly the seat of the lesion or subjected to the pressure of a vertebral tumor, and twitchings of the muscles, and contractions of the limbs, if the antero-lateral columns are principally involved. When both sets of columns—as is generally the case—are affected, the troubles of sensibility and of motility are both present.

If the tumor is situated in the cervical or upper dorsal region, there is generally tonic contraction of the muscles of the neck by which the head is thrown backward, causing the patient to present the appearance of a person affected with the opisthotonos of tetanus. There are in such a case usually ocular troubles, such as those previously mentioned, and more or less gastric derangement. The symptoms, so far as the limbs and viscera are concerned, vary in their extent according to the situation of the morbid growth.

The symptoms of strong compression are anæsthesia and motor paralysis. These may or may not be accompanied with muscular atrophy. Reflex excitability and electro-muscular contractility are generally at first increased, or at least not lessened, but, as the pressure augments and the structure of the cord becomes more disorganized, they are lessened.

The bladder generally retains its power, but if the tumor be situated so as to compress the middle of the dorsal region there will be more or less difficulty in passing the urine which is retained through spasm of the sphincter. If the lesion exists at the upper part of the lumbar region, or at about that part, the bladder and sphincter will be paralyzed, and the urine will dribble continuously.[1]

Many cases, of what may with Drs. Charcot and Brown-Séquard be called hemi-paraplegia, are due to spinal tumors. It often happens that these are small and compress a lateral half of the cord, leaving the other affected only by the transmitted pressure. A very remarkable case has been reported by Charcot,[2] in which the left inferior extremity was completely paralyzed, while the right was simply weak without having lost the power of contraction in any of its muscles. On the other hand, sensibility was greatly lessened in the right limb, while it was exalted in the left. There was paralysis of the bladder, but no atrophy of either limb. Finally, anasarca and bed-sores appeared, and the patient gradually sank. On post-mortem examination, a tumor was found growing from the dura mater on the anterior face of the cord and compressing its left lateral half. The accompanying woodcuts (Figs. 91 and 92), reduced from Charcot's lithographic representations, show the situation and relations of this tumor. Fig. 91 shows the growth *in situ*, and Fig. 92 the parts as they appeared when the tumor was pushed aside so as to allow the cavity to be seen in which it was lodged.[3]

Recollecting the facts that the fibres of the anterior or motor columns of the cord decussate at the medulla oblongata, while those of the posterior or sensory columns cross over soon after they enter the cord from the posterior roots of the spinal nerves, we can understand why, when the paralysis of motion is confined to one side, or is greater on that side, the lesion is on the corresponding side of the cord, and this loss of motility should be accompanied with anæsthesia of the opposite side of the body.

Under the name of painful paraplegia (*paraplégie douloureuse*), Cruveilhier referred to a form of spinal disease which has been subsequently described more fully by Charcot. This latter author has observed six cases, in all of which there was cancer of the mammary gland. In three of these he had the opportunity of making post-mortem examinations, and discovered carcinoma of a lumbar vertebra in each, to which the irritation and compression of the cord were due. According to him, "the skin, especially during the paroxysms of pain, is often very sensitive to the touch. At the same time walking becomes troublesome, and later the patient cannot walk without help; finally, muscular atrophy ensues, and the patient loses the power to stand."

---

[1] Charcot, "Leçons sur les maladies du système nerveux ; seconde fascicule. De la compression lente de la moëlle épinière," Paris, 1873, p. 114.

[2] *Archives de physiologie*, No. 2, p. 291.

[3] This case is quoted at length by Dr. Brown-Séquard in the *Lancet* of September 25, 1869, p. 429. In previous and subsequent numbers of this journal Brown-Séquard has contributed much valuable information on the subject of hemi-paraplegia.

FIG. 91.

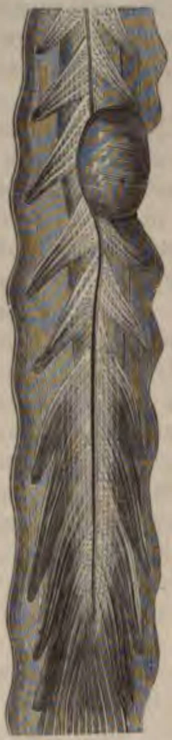
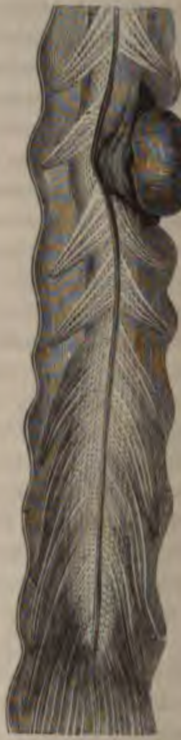


FIG. 92.



Simon, from whom I quote these details, under the head of "paraplegia dolorosa," describes a case which came under his own observation, in which, during life, symptoms similar to those mentioned by Charcot were noticed, and in which, after death, a cancerous tumor was found growing from the first lumbar vertebra and compressing the posterior columns of the cord. Other lesions were present in the posterior columns both above and below the tumor; they were apparently of the nature of sclerosis. Similar cases have been described by other authors.

Although it is rendered certain that cancerous tumors of the vertebræ may give rise to paraplegia characterized by great pain, it must be borne in mind that these symptoms are not a necessary accompaniment of the lesion, and that they are met with in other affections of the cord.

A tumor situated in the cervical or upper dorsal region of the cord sometimes gives rise to characteristic symptoms. Thus there may be dilatation or contraction of the pupil on one or both sides, or one may be contracted and the other dilated. Cough and dyspnœa, vomiting, difficulty of swallowing, epileptiform convulsions, and a remarkable slowness of the pulse, are sometimes among the phenomena. But such symptoms are by no means invariable. Many years ago Velpeau[1] reported a case of tumor of the cervical region of the cord in which none of these symptoms were present. The patient, a woman at thirty-four years of age, after having experienced mental troubles and been exposed to bad hygienic influences, suffered from convulsive movements of the limbs which were not of long continuance. Shortly afterward the left arm became the seat of a severe pain, and she had pains in the head. The pain in the arm increased, and little by little she lost the use of the limb. Renewed convulsive movements occurred in the inferior extremities, and were followed by complete paralysis. When she presented herself at the hospital she had no pain in the left arm, which was, however, entirely paralyzed, but which, nevertheless, retained its sensibility almost unaltered. Motion of the right arm, though difficult, was still possible, but it was the seat of very severe pain. Respiration was normal but a little weak; the pulse was frequent, sometimes strong, but generally small and regular. There was a large and deep ulceration on the sacrum; the lower extremities were anasarcous and were deprived of all sensation and power of motion. The fecal matters and the urine were passed involuntarily and unconsciously. Gradually she lost the ability to move the right upper extremity. She sank almost imperceptibly without apparent cause, and died after having been two months and a half in the hospital.

On post-mortem examination numerous whitish opaline plates were found scattered over the arachnoid, but the principal lesion consisted of a tumor, which was situated between the arachnoid and the cord, and covered the entire anterior surface of the latter from the sixth cervical pair of nerves to the third dorsal. This growth appeared to have its origin in the left antero-lateral furrow. The anterior roots of the left spinal nerves within its area were so compressed that they were shrunk to mere threads, and the posterior roots of the same side were also subjected to pressure. The right posterior roots were in a normal condition. The whole body of the cord was flattened by this tumor, but the left side was especially in this condition. The growth was cerebriform in appearance, and was thought to be cancerous.

As an example of the symptoms resulting from a tumor occupying the dorsal region of the cord, the following, from Ollivier,[2] is cited:

---

[1] "Observation sur une maladie de la moëlle épinière tendant à démontrer l'isolement des fonctions des racines sensitives et motrices des nerfs," *Journal de physiologie* de Magendie, tome vi., 1826, p. 138.

[2] "Traité des maladies de la moëlle épinière," Paris, 1837, tome ii., p. 477.

A woman, aged fifty-two, had enjoyed good health till in 1819 she began to experience lancinating pains in the abdomen and breast. After several months these pains shifted their situation to the pelvis and the lower extremities, especially the left. These limbs then became the seat of varied phenomena, sometimes being cold, at others hot, and again numb; they were also subject to the most intolerable itching. Then they became by turns immovable, and were agitated by convulsive movements. Although she could stand, walking was impossible. Finally, in February, 1821, they began to atrophy, and at once lost all sensibility and power of motion. Then these symptoms disappeared, and there only remained numbness and pains apparently starting from the pelvis and traversing the nerves. In May, 1821, she entered the hospital. At this time the inferior extremities were rigid, and could not be flexed without causing pain of a very atrocious character. They were insensible to all external excitations, but were constantly the seat of severe and lancinating pains. There was, however, no pain along the vertebral column, and the general health of the patient was excellent.

All these symptoms persisted till in January, 1823, the legs began to be flexed on the thighs, and these latter on the pelvis, to such an extent that the heels pressed against the buttocks, and the knees touched the chest. Forced extension of the limbs was exceedingly painful, and when they were by main strength extended they at once returned to their former position as soon as the traction was discontinued. Two months before her death the left wrist and right knee became inflamed; the former suppurated, and the patient died six weeks afterward. Strychnia had been administered, but always aggravated the symptoms. Morphia gave no relief.

Examination after death showed the brain to be healthy. There was a band of sclerosed tissue on each side of the cerebellum.

The spinal cord was healthy as far down as the tenth dorsal vertebra. Here a tumor existed between the two layers of the arachnoid. The growth was oblong, and about two inches in length. It was similar in appearance to brain-tissue, but firmer. It was not adherent to the cord, but throughout its whole extent pressed on the organ, which was softened throughout to the consistence of a thin jelly. At the most voluminous part of the tumor the cord was so much compressed that it was almost cut in two, so that there was the appearance of two cones with their apices together. A careful examination showed that in the softened part no trace of nerve-structure remained.

Leyden,[1] among other interesting cases, gives the following, of tumor occupying the lower dorsal region of the cord:

The patient, a woman twenty-nine years old, after being delivered of a dead child, became affected with a pain in the right leg, which, starting from the foot, reached the knee, and finally settled in the calf. She noticed at the same time a weakness of this leg, which prevented her walking well, and eventually confined her to bed. These symptoms disappeared, and she remained well for over three years, when the right leg again became weak, and was the seat of constant lancinating pains, which were aggravated by muscular exercise. In April, 1872, the left leg was also affected with similar pains. It soon became impossible for her to bend the knee or to move the limb. All these symptoms increased until, in February, 1872, she was unable to walk, and there was complete anæsthesia in both extremities as high as the hips. A painful sensation of constriction was felt around the body at the umbilicus. The electric excitability of the right lower extremity was lessened, of the left was normal. The reflex excitability of both lower extremities was increased; the nutrition was good. At times they were the seat of strong contractions.

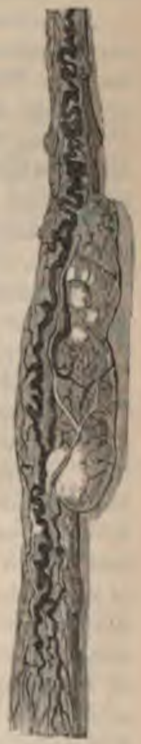
---

[1] "Klinik der Rückenmarkskrankheiten," erster Band, Berlin, 1874, p. 454.

By August, 1873, the patient was entirely confined to bed on her back, and deprived of all voluntary movement of her lower extremities. There were, however, often paroxysms of tremor in both feet so strong as to shake the whole body, and at times powerful contractions of the muscles, drawing the thighs against the abdomen, while the knees were flexed to their utmost extent. The constricting pain around the body was still present.

In the beginning of October the patient was seized with typhus fever and died. On examining the spinal cord it was found that a tumor existed on the right side, reaching from the seventh to the tenth dorsal vertebra, and firmly attached to the dura mater. The entire length of this growth was eighty millimetres (a little over three inches). (Fig. 93.)

FIG. 93.



Causes.—Nothing is known relative to the etiology of intra-spinal tumors beyond the fact that they may result from the syphilitic, scrofulous, and cancerous diatheses, and from wounds and injuries.

Diagnosis.—There are no certain marks by which we can determine with any great degree of certainty that a tumor is compressing the spinal cord. We may suspect such to be the case when the motor paralysis is more marked on one side of the body than the other, and the anæsthesia exists to a greater extent on the opposite side. The existence of either syphilis, scrofula, or cancer, in connection with spinal troubles not clearly referable to some other disease, may likewise excite the suspicion that a tumor exists. But the symptoms—paralysis, hyperæsthesia, anæsthesia, contractions, rigidity, and spinal convulsions—are met with in other spinal disorders, notably in symmetrical lateral sclerosis. The unilateral predominance of the phenomena is probably, on the whole, most to be relied upon as a diagnostic mark.

Prognosis.—This is always unfavorable. It is less so when a syphilitic origin can be made out. No others recover.

Morbid Anatomy and Pathology.—The most common intra-spinal morbid growths are those which are developed from the vertebræ, and they include many syphilitic, scrofulous, and cancerous tumors. They originate either from the bones or from the periosteum. Formations resulting from either of these diatheses may also grow from the meninges or the substance of the cord.

Parasitic tumors due to either the echinococcus or the cysticercus, may also be developed within the spinal canal. Their usual seat is in the membranes; and, according to Ollivier,[1] the echinococcus is found in the spinal cavity of women only.

Aneurismal tumors occasionally form in the intra-spinal arteries, and may compress the cord. Aneurisms of the thoracic or abdominal aorta may, by pressure, cause absorption of the vertebræ, and may thus eventually subject the cord to their influence.

Among the other intra-spinal tumors are the glioma—a growth, the seat of which is especially in the brain and spinal cord, and whose structure is very similar to that of sclerosed nerve-tissue—the sarcoma, the psammoma, the neuroma, fibroma, and myxoma, and tumors, generally syphilitic, developed from the vertebræ.

Treatment.—The attempt should always be made, whenever the existence of a tumor of the spinal cord is suspected, to effect its removal by anti-syphilitic treatment, with iodide of potassium and mercury. The following case will show the advantages of following this course:

In the summer of 1869 I was requested to visit a gentleman who, I was informed, was paraplegic and subject to paroxysms of great suffering. On making my examination, I found his limbs contracted, his reflex excitability augmented, and motor paralysis and anæsthesia of both lower extremities. There were intense pain in the lower dorsal region, and violent spasms of the sphincter vesicæ, alternating with paralysis of it and the bladder. There were also paroxysms of severe pain in the head, and occasional attacks of delirium. He denied any syphilitic infection, but, on examining his head with my hands, I found a gummy tumor of the scalp over the right occipital region. Further inquiry and examination revealed the existence of a similar tumor over the left radius. I inferred that there might be one or more like growths within the spinal canal, and I administered the iodide of potassium in gradually-increasing doses, with the bichloride of mercury in doses of the sixteenth of a grain three times a day. In less than two months every symptom of disease, except a general weakness, had disappeared. The tumor of the scalp went during the first month; that of the arm a week later. The iodide of potassium was carried up to fifty grains three times a day. This patient continues in good health up to the present time. Even if there was not sufficient reason to diagnosticate the existence of an intra-spinal syphilitic tumor, the success of the treatment can scarcely leave a doubt on the subject.

---

[1] "Traité des maladies de la moëlle épinière," Paris, 1837, tome ii., p. 549.

If this treatment fail, there is nothing else left. As Leyden says, the only means of removal consists in extirpation by trephining the vertebral column. Who will be the first to attempt the operation?

As means of mitigating the pain and spinal convulsions, hypodermic injections of morphia or atropia, or of both combined, may be employed.

CHAPTER VIII.

SYPHILIS OF THE SPINAL CORD AND ITS MEMBRANES.

When compared with like affections of the brain and its membranes, syphilitic accidents of the cord and its envelopes are certainly rare. Of course, this statement has reference only to new formations. As a cause of many of the affections described in the foregoing chapters, syphilis occupies, if not the first place, at least one very near the front rank. Locomotor ataxia, for instance, is probably in the majority of instances of syphilitic origin; and Dr. Gowers has recently gone so far as to declare that in his opinion syphilis is its only cause.

As in the brain, neoplasms of syphilitic origin are known to be developed on the periphery of the cord rather than in its substance, in the subarachnoid space and on the internal face of the dura mater. Adhesions of the membranes to each other and to the substance of the cord are thus induced, while this latter is little by little invaded by the new formation. Generally the neoplasm does not appear as a well-defined tumor, but as a substance analogous to that of gummata diffused through the tissues.

The histological and macroscopic characters are like those which are met with in like formations of the encephalon. Instead of a diffused infiltration, little miliary nodosities may be met with, disseminated in the meninges. Engelstedt[1] has reported a case of this kind.

Sometimes there is found at the *post-mortem* examination of syphilitic patients, who had during life presented evidences of spinal troubles, a kind of deposit replacing to a certain extent the cellulo-adipose tissue which lines the internal face of the spinal canal. From this there results an intimate adherence of the dura mater with the walls of this canal. Virchow has reported a similar case observed in an individual who had had multiple syphilitic accidents, and in the last period of his existence a painful rigidity of the neck and arms, which supervened on paralysis of the upper extremities. At the autopsy the dura mater was found considerably thickened at the height of the fifth and sixth cervical vertebræ, and adherent to the wall of the canal by a great quantity of tough connective tissue.

---

[1] "Archiv der Heilkunde," B. iv., 1863, p. 169.

At other times the exudation occupies the internal face of the dura mater, and this results in adhesions of the membranes to each other. At the same time the adjacent part of the cord is the seat of a hyperplasia of the neuroglia with distention of the nerve-structures. Hueter reports a case of this kind.

In cases of syphilitic patients who have died after having presented symptoms of a spinal affection, a simple softening of the cord has been discovered. On the other hand, there have never been any absolute proofs that pure myelitis has ever been developed through the influence of syphilis.

And in a certain number of cases the autopsy has never revealed the slightest appreciable lesion of the cord (Zambaco, Kussmaul, Leon, Gros, and Lancereaux). Spinal affections are generally exhibited at an advanced period of syphilis, and in individuals who present undeniable traces of the diathesis with all the accompaniments of a more or less advanced cachexia. They are characterized by pains localized in the spine or radiating to the limbs, with various derangements of sensibility (formication, numbness, anæsthesia, etc.). Little by little rigidity of the muscles supervenes, and this is succeeded by temporary contractions and movements which gradually lose their energy, and are accompanied with painful cramps. All these symptoms, which are generally regarded as being due to meningitis, are subject to alternations of amelioration and aggravation, and eventually all phenomena of excitation give place to paralysis. This generally first shows itself in one of the lower extremities, and advances with great rapidity. Very soon the opposite limb is attacked, and the paraplegia becomes complete. Often the sphincters alone are involved. It is to be noticed that the paralysis of sensibility does not keep pace with that of motion, which, after existing for a long time, is supplemented by phenomena of anæsthesia or parathesia. Then there is often a period of repose. At this period proper treatment may procure for the patient a gradual but nevertheless satisfactory cure. This termination is, above all, to be looked for when the lesion remains confined to the inferior part of the cord. The prognosis is much less favorable when the genito-urinary functions are involved. In such cases we ordinarily find that sooner or later cutaneous trophic troubles are developed over

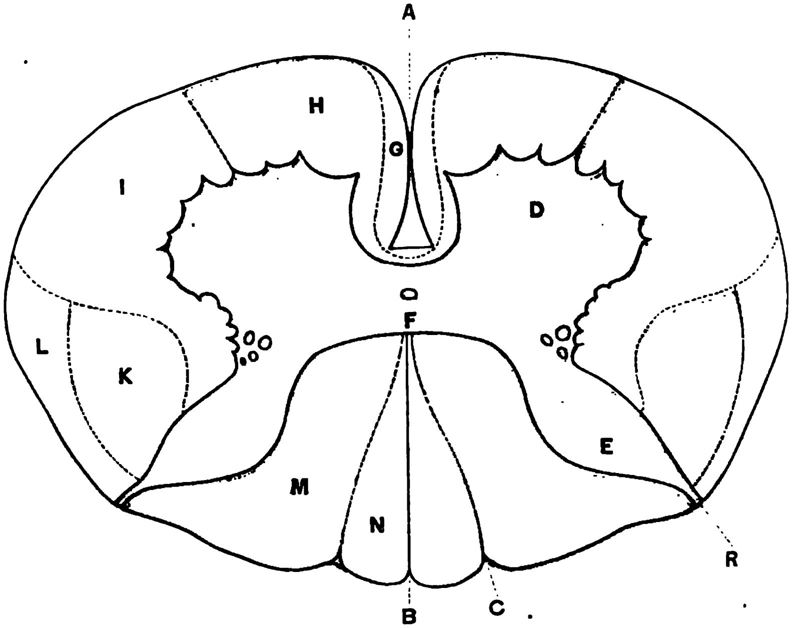


Diagram illustrating the relations of the nerve-fibre tracts in the spinal cord. The section is supposed to be taken transversely through the lower part of the cervical enlargement (slightly modified from Flechsig):

A. Anterior Median Fissure.
B. Posterior Median Fissure.
C. Intermediate Fissure.
D. Anterior Gray Cornu.
E. Posterior Gray Cornu.
F. Gray Commissure, with Central Canal.
G. Uncrossed Pyramidal Tract (Flechsig), or Column of Türck.
H. Fundamental Part of the Anterior Column (Anterior Root-Zones of Charcot and his pupils).
I. Anterior Part of Lateral Column.
K. Crossed Pyramidal Tract of Lateral Column.
L. Direct Tract from Lateral Column to Cerebellum.
M. Column of Burdach, Posterior Root-Zones of Charcot and his pupils.
N. Column of Goll.

The posterior columns of descriptive anatomy include the fields M and N extending on the surface from B to R. The antero-lateral columns extend on the surface from R to A. Their anterior division includes the fields G and H; their lateral division, the fields K, L, and I.

the sacrum—purulent cystitis, etc.—with all their consequences. Hectic fever is excited, and the patient dies greatly emaciated.

When syphilitic lesions affect the upper part of the cervical region of the cord, patients are exposed to still greater dangers. In such cases the symptoms are very rapidly developed, for the paralysis involves all the muscles of the trunk, including the respiratory apparatus. The disease, in fact, follows a course analogous to that of acute ascending paralysis. In such instances a specific treatment instituted opportunely may still be sufficient to save the life of the patient, but cannot effect a complete cure. The tissues which have been infiltrated with the syphilitic exudations undergo a veritable inodular retraction and alteration, to which there is sometimes added secondary ascending and descending degenerations. When the secondary degeneration affects the posterior columns, the paralysis may be replaced by certain manifestations of tabes dorsalis, but this syphilitic ataxia is not to be confounded with ordinary locomotor ataxia.

In the cases of those patients in whom at the autopsy appreciable alterations of the marrow are not found, the spinal affection has generally followed a subacute course. It then greatly resembles the acute ascending paralysis of Landry. Sometimes its real character is recognized as being like that which is exhibited at an early period of syphilis, in the course of the first year after contamination. Ordinarily it is not preceded by any prodromatic symptoms, but it is sometimes the case that a very short time after the development of the paralysis the affected limbs are the seat of vague pains. The paralysis begins in the lower extremities, and is complete after a very few days. It is accompanied by a certain degree of weakness of the bladder, which is manifested either by incontinence or retention of urine. After the second week the patient is confined to his bed, and in a period relatively short he succumbs to septic infection. Therapeutics can avail nothing against this form of syphilitic myelitis.

This concludes what I have to say relative to the diseases of the spinal cord. I have endeavored to make the subject as plain as possible, but, in the study of a class of diseases still to a great extent obscure in their medical relations, there must necessarily be defects in the description.

In order to a better understanding of the normal and morbid anatomy of the cord, as established by the most recent investigations, I have enlarged and modified from Flechsig a diagram of a transverse section, which will be found to give, on examination, very exact information. In it are clearly indicated the several divisions of the cord with the study of which we have been engaged.

SECTION III.

CEREBRO-SPINAL DISEASES

CHAPTER I.

HYDROPHOBIA.

ALTHOUGH there are objections to the name employed to designate the terrible disease I now propose to consider, the same is true of all other terms which have been applied to it, and the present has the advantage of being well known. So long as we are obliged, through ignorance of pathology and morbid anatomy, to use a nomenclature based on symptoms, we must expect to be inexact. The name hydrophobia is as old as Galen, and still retains its preëminence, notwithstanding the fact that the symptom on which it is based is sometimes absent.

Symptoms.—Beginning with the reception of the injury by which the body has been inoculated, we find that it heals in the ordinary way, and that there are no immediate signs of infection. At a period which varies greatly in different cases, pain or a sensation of uneasiness is usually experienced at the seat of the wound. This, however, is rarely of such intensity as to cause suffering, and probably would generally be overlooked or disregarded but for the apprehension which the patient has, and which directs his attention to every sensation which can be attributed to the wound. But there may be absolutely no pain or uneasiness other than such as are met with in all wounds till the phenomena of the affection are manifested. The period between the reception of the injury and the beginning of the symptoms of hydrophobia is known as the stage of incubation.

The duration of this stage is variable. It is rarely shorter than a month, and probably never longer than two years. Instances are on record, however, in which the disease has been developed within ten days, and others, about which, however, there is much doubt, in which the latent period has reached to ten years and longer. The vast majority of cases occur within seven months after the reception of the wound. In six cases which have been under my observation, the period of incubation varied from about twenty-five days to four months and a half.

Dr. John Johnston,[1] however, refers to an opinion that in hot countries the disease has appeared in four or five days after the bite, and, on the margin of the page on which this statement is made, Dr. Hosack, to whom the book formerly belonged, has written a note, in which he states that it ensued in a child in New York five days after the bite was inflicted.

During this period of incubation there are not often any indications of what is going to take place except in those cases in which there are abnormal sensations in the cicatrix or its neighborhood. Sometimes there are depression of spirits, anxiety, and derangement of the digestive functions, but these symptoms may fairly be attributed to the peculiar circumstances of the case, aside from any toxic influence due to infection.

The first symptoms which appear are often directly connected with the cicatrix, which, if it has previously been free from abnormal appearances and sensations, now becomes subject to both. But there is no constancy even in these phenomena. They were altogether absent in one of my cases, and very slightly manifested in one other, if they were present at all. In this case, which I saw in consultation with Dr. S. G. Cook,[2] of this city, the patient, after other symptoms had appeared, occasionally clutched the place where he had been bitten, but denied, on being asked, that there was any pain at the spot.

But, though there may be no symptoms of swelling, redness, or pain about the cicatrix, there are abnormal sensations in the nerves which radiate from it. Thus, if the injury has been in the leg, pains are felt along the courses of the sciatic and crural nerves ; if in the hand, similar sensations are experienced in the radial, ulnar, median, and other nerves of the upper extremity. Occasionally the pain is felt in the epigastric region, and in any situation is ordinarily accompanied by headache. At about the same time the respiration becomes sighing and irregular, there is a feeling of oppression or constriction in the chest, the pulse loses its force and uniformity, and there is an indefinable sense of anxiety. The sleep is scarcely ever natural. Either there is insomnia or drowsiness, and sleep, when obtained, is disturbed by frightful dreams, and is unrefreshing. The bowels are constipated, the skin is dry, and there are alternate chills and flushes of heat. The duration of this stage is from two to four days.

And then the period of full development begins ; characterized, at first, by an increase in the symptoms just mentioned, and subsequently by the appearance of others not previously present. A peculiar sense of uneasiness is felt at the epigastrium, and a pain and constriction of the throat, which add greatly to the distress. The tongue becomes stiff and painful, and articulation is thereby rendered indistinct; the respiration increases in irregularity, and becomes noisy and oppressed; the rigidity of the muscles of the throat prevents or impedes deglutition, and the patient dreads attempting to swallow, from the experience he soon acquires that his efforts in this direction are attended with pain and spasm, which greatly increase his sufferings. Sometimes the convulsion of the pharyngeal muscles is so great that substances are thrown with great force out of the mouth. This was the case in three of the instances I witnessed. At the same time the spasm extends to other parts of the body, and occasionally becomes general. It is accompanied by pain in the epigastrium, and sometimes in the spine. Solids are swallowed with much more ease than liquids. Indeed, so great is the difference that the patient cannot even entertain the idea of swallowing any fluid, without being thrown into spasms. The sound of water splashing or trickling, the sight of it, the thought of it, and even an impression remotely connected with water, such as that produced by the reflection of rays of sunlight on the face by a mirror, will bring on a paroxysm of convulsions. With the spasm there are sobbings, trembling, and then a condition of exhaustion, during which the patient is bathed in perspiration.

---

[1] "Cases of Lyssa, with Remarks," p. 5, in "Medical Essays," printed 1795 to 1805.

[2] "A Case of Hydrophobia," *Journal of Psychological Medicine*, January, 1870, p. 80.

The following day the phenomena are still more strongly marked. The mouth is dry and parched, and yet the patient dare not attempt to quench his thirst; vomiting ensues, the pulse becomes rapid and small, the pain in the pit of the stomach still increases, the headache is intense, and the countenance expresses terror, anxiety, and suffering. The pain in the spine augments and extends to the muscles of the neck and abdomen. The secretions of the mouth are altered, and the saliva is mixed with a frothy, tenacious mucus, which the patient is constantly attempting to eject, but which collects as fast as he can spit it out. The mouth and fauces are dry and painful, articulation is almost impossible, and every attempt to relieve the distress by a few drops of water induces a return of the spasms and convulsions. Finally, every reflex excitation reaches the muscles of the throat; the contact of the bedclothes, the jarring of the bed by persons walking in the room, the rustling of window-curtains—any thing capable of acting on the hearing, the eyesight, or the touch, may cause the spasms.

As the disease advances, all the symptoms increase in violence, and still others make their appearance. The urine and fæces are often passed involuntarily, the skin becomes exquisitely sensitive, the body is in a constant state of agitation and tremor, alternating with spasms, and the tough, stringy, tenacious mucus collects in the throat and impedes respiration.

Thus far the mental symptoms have scarcely been considered, but they are present almost from the first. Indeed, they may be among the very first indications of disorder. They consist of emotional disturbances of various kinds, and sometimes radical changes of character and disposition.

It has been alleged by some authors that the dreams, at a very early period after inoculation, are connected with the animal giving the the wound. I have never met with this symptom, but in the case previously cited, and which I saw twice in consultation, a circumstance still more remarkable is related by Dr. Cook. The patient, a child three years old, was bitten by a bitch in heat on or about August 20, 1870. On November 15th the mother noticed that he slept badly; on the 16th, among other manifestations, he "was cranky all day." On the 17th he was seen by Dr. Cook.[1]

"On entering the room," says the doctor, in his report of the case, "and seeing several children, and not noticing any thing wrong with any of them, I very naturally inquired which was the patient. I was pointed to a little boy sitting at a table in a high chair. On approaching him, he turned his face toward me, revealing the most peculiar-looking eyes I have ever seen. They were not like those seen in persons suffering from delirium in prolonged fevers, nor yet like those we see in the second stage of cerebral meningitis, although somewhat resembling both of these conditions, but more like the eyes of a person in a fit of violent anger, slightly combined with a feeling of fear.

"When I reached out my hand to touch his, he shrank from me as from a blow, at the same time making a desperate effort to catch his breath, precisely as a naked person might if a pail of cold water was unexpectedly poured over him. This I understood to be a laryngeal spasm. It was very brief, lasting but the fraction of a minute, probably not more than ten seconds. I took a seat a little distance from him, where I could see his every motion, and regarded him attentively for a long time.

"He seemed an unusually intelligent child, for one of his age, speaking very distinctly with a clear, ringing voice, which his parents informed me was a little unnatural, as it 'seemed strained.' He had at times a disposition to stammer, which was also unnatural. For one hour after my observation commenced, he talked almost incessantly of dogs, and repeated very few sentences a second time. He seemed familiar with all the most common breeds, relating some anecdote of the bull-dog, the mastiff, the bird-dog, the spaniel, the coach-dog, and the poodle.

"Connected with all his naratives was a tragic or gloomy termination. The mastiff, after carrying him an incredible distance about the city, finally disappeared through a bottomless hole in the street, he only escaping a similar fate by suddenly dismounting. The bull-dog, after bringing for his admiration and pleasure a great variety of puppies, suddenly turned cannibal, and swallowed the whole lot. The spaniel, after having been his playmate for a very long time, finally took it into his head one day to get on to a coffin that was being carried through the streets, and ride away to reappear no more."

---

[1] *Op. cit.*, p. 81.

There were no other evidences of disordered mental action in this child, and he died, perfectly conscious to the last.

Usually, however, this is not the case, and various morbid desires are entertained by the patient. Thus, in a case which I saw in this city in 1865, there was an impulse to strike those near, and an intense dislike of certain persons who had always been intimate friends of the patient. In both the other cases there were paroxysms of previous delirium, during which the sufferers bit and struck at all within their reach, and of which hallucinations and delusions constituted marked features. In the case of the boy just cited, the stories of dogs which he related were evidently delusions which he accepted as realities.

The temperature is always elevated from the very beginning of the disease. It is rarely below 105° Fahr., and may rise as high as 110° during the height of a paroxysm or immediately after its cessation.

Death usually takes place on the third day after the accession of the symptoms indicating the full development of the disease. The chief of these is laryngeal spasm. A fatal termination is rarely delayed till after the third day, though cases are not uncommon in which it has ensued on the first or second day. In all the cases, except two, which have been under my observation, the third was the fatal day. In Dr. Cook's the disease may be considered as having been fairly developed on the 17th of November, the first day in which any spasm of the throat was witnessed. Death resulted on the evening of the 18th.

In June, 1874, I attended, in consultation with Dr. Alexander Hadden, my sixth case of hydrophobia. The patient, a man about twenty-five years of age, had been bitten about three weeks before by a dog not clearly identified. When Dr. Hadden first saw him on the 24th of June, at 8.30 P. M., the man was in bed, complaining of nervousness, soreness in his neck and throat, and a strange feeling of tightness around the chest. His countenance was anxious, his pupils were dilated, and his general appearance was that of a person facing some impending danger, and not in extreme pain. He said his throat was sore, and that he could not swallow any thing, not even water. Examination showed that there was no congestion or inflammation of his throat. His pulse, respiration, and temperature, were normal, excepting that he occasionally sighed. There was also a little disposition to hack and spit. He complained of thirst, but said he knew he could not drink, for the very sight of water made him shudder. He was told to try, and some water was brought, but the sight of it caused a violent spasm. He threw himself around in the bed backward and forward, and ordered the water to be taken away. He immediately afterward called for the goblet, said he was thirsty and must drink, seized it, and with a violent effort succeeded in taking a single swallow, which was followed by a severe convulsive shudder and contraction of the muscles of the neck and chest.

Dr. Hadden, recognizing the symptoms of hydrophobia, asked if he had been recently injured by any animal. At first he replied in the negative, but on the doctor's saying, "Not by a dog?" he answered, "Only slightly on the knuckle of the right hand by a little black dog belonging to a baker around the corner on the avenue." He further stated that there was nothing the matter with the dog, for he had seen it afterward, and only about a week since it had been taken to the pound and the bite was inflicted three or four weeks before.

For two days previously he had felt badly, was thirsty, and had drunk a good deal of water; and the evening before had gone out, but soon returned, saying he felt chilly. While taking a cup of tea at 6 P. M. that day (the 23d), he had experienced the first difficulty in swallowing. Shortly afterward, while going to the kitchen, a cool draught of air blew on him and caused him to stagger so that he nearly fell.

The next morning Dr. Hadden saw him with Dr. Leavitt.

"We found him in a frightful state of excitement; had broken down the bed, and was struggling with his attendants to get at liberty. He was shouting and crying out to them to let him go, and called for water, which, when brought, he could not drink. His mind was clear, and he knew all those around him; was spitting a viscid saliva, and was careful not to spit on any one, not even on his clothes. It was so abundant that his attendants were obliged to wipe it from his lips. Dr. Leavitt and myself, after viewing the case in all its aspects, concluded to inject in the tissue of the leg one-half a grain of morphine and one sixty-fourth of a grain of atropine in solution, which was done at 3 A. M. by Dr. Leavitt. We carefully watched the effect till 3.30 A. M., when his violence having in no way abated, another injection was given in the same part, of three-eighths of a grain of morphine and one-eighth of a grain of atropine, which in some degree produced the characteristic effect of morphine, and very clearly the appearances of the atropine; for, notwithstanding he was struggling violently, the saliva, which had been very troublesome, was completely dried up, so much so that the patient himself remarked that he was very thirsty, and his mouth felt as if he had been chewing a brick. Fifteen drops of chloroform were then injected, with no effects whatever, unless to weaken his already weak and frequent pulse. At 4.15 A. M., three-eighths of a grain of morphine were again introduced under the skin, without atropine. This quieted the patient so that he was easily restrained, and he remained in this condition from 4.30 A. M. till 10 A. M., when the effects had so far passed off that the attendants were alarmed at his violence, and the abundance of saliva he was spitting from his mouth. Dr. William A. Hammond saw him with me at this time. He supported the diagnosis and thought well of the treatment; he saw that it subdued violence and suppressed the flow of saliva—the two most important features of this hopeless disease. At 10.15 A. M., by his order, three-eighths of a grain of morphia in solution were injected into the tissue of the thigh, which served to temper down the increasing violence of the spasms, but did not stop the flow of saliva. I accordingly, at 10.45 A. M., injected three-eighths of a grain of morphia and one-fortieth of a grain of atropia, which had the desired effect of producing the quieting effect of the morphia as well as the specific effect of the atropia on the salivary glands. The poisonous effects of the morphia and atropia were at no time apparent. He died at 4.15 P. M., June 26, 1874, about twenty-four hours after the first spasm."[1]

As stated by Dr. Hadden, I was called to see the patient at about ten o'clock on the morning of June 26th. When I went into the room he was lying upon the floor pinioned, to a certain extent, and surrounded with pillows to prevent him injuring himself. He was then spitting continually; in fact, every expiration was accompanied by an effort to spit out the thick, tenacious mucus so characteristic of hydrophobia. His pupils were largely dilated, but, as Dr. Hadden stated, not more so than before the atropia was given. He was able to converse with tolerable fluency, and, when I put two or three questions to him, he answered, but not very directly. So far as I could make out, both from his answers and appearance, he was not suffering from acute pain. There was a good deal of movement of his limbs, not apparently spasmodic, for there seemed to be the element of volition in the actions he made with his arms and legs. He could not swallow fluids, and even a piece of ice given to him was ejected with force from his throat. His pulse was too rapid to be counted, and his respiration was hurried and irregular.

I fully concurred in the suggestion to give him morphia for the purpose of moderating the intensity of his symptoms. A hypodermic injection was administered, and a sedative effect was produced. After I left, his paroxysms returned with great violence, and he died that afternoon.

Generally death occurs during a spasm. This was the result in four of the six cases I have witnessed. In the others the patients died quietly, a consequence probably of the sedative medicines administered. When death takes place during the former condition it is probably due to apnœa; in the latter, to exhaustion. In all cases the powers of life, from the violent convulsions, the loss of sleep, and the deprivation of food, are drained away to their utmost.

---

[1] "Report of a Case of Hydrophobia," by Alexander Hadden, M. D., "Proceedings of the New York Neurological Society," *Psychological and Medico-Legal Journal*, September, 1874, p. 166.

Causes.—It has generally been supposed that hydrophobia has but one source in the human subject, and that is inoculation by the saliva of an animal affected with rabies; that it cannot be communicated to one individual of the human species by the saliva of another affected with hydrophobia; and that neither dogs nor other animals can be infected by inoculation with the saliva of a hydrophobic man. Magendie's experiment as to the latter point is considered by many to be of exceedingly doubtful value, as hydrophobia was, it is said, prevailing among dogs at the time, and that hence the animal may have been bitten. As we shall see hereafter, these suppositions are all more or less ill-founded.

Thus it is very probable that the saliva of healthy animals, the dog especially, is, under certain circumstances, capable of producing hydrophobia in man and other animals. A case of the kind is recorded in *Hufeland's Journal* of December, 1839, and similar ones are frequently met with. In none of the cases I have witnessed was the dog which had inflicted the wound supposed to have been rabid. In one case which I saw in this city, with a physician whose name I cannot recall, the patient, a stableman, was bitten by a dog that was to all appearance in perfect health. In the case reported by Dr. Cook, the animal, a bitch, was being led quietly through the passage-way of the house, when the child became entangled in the chain, fell against the dog, and was bitten apparently in anger. The animal was well known, and was not even suspected of being hydrophobic. She was in heat; and Dr. Cook raises, for the first time to my knowledge, the question whether this circumstance renders the saliva of the animal capable of inducing hydrophobia in the human subject. With a view of throwing as much light as possible on the subject, he consulted the records of Bellevue Hospital, in order to ascertain the facts in relation to a man who died of what was supposed to be hydrophobia from the bite of a bitch in heat. The result of his inquiries was to show very certainly that the man did die of hydrophobia; that the animal was not rabid, and that she was in heat.

In the case, the details of which have just been given, there was a good deal of doubt in regard to the identification of the dog which inflicted the bite. The patient said he had been bitten "by a little black dog belonging to a baker around the corner on the avenue." But no such dog was known, and there was no baker "around the corner," on either Second or Third Avenue. The only dog that was known to have bitten the man was alive and well on the 7th of July, two weeks afterward.

---

[1] "Dictionnaire des sciences médicales," article "Rage," tome xlvii., p. 46. Also *Journal de physiologie*, tome i., p. 47.

In the present state of our knowledge, it is useless to pursue this point of the inquiry further. It is one in regard to which certainty appears to be impossible of attainment. Fleming,[1] however, seems to admit the possibility of an animal under strong sexual excitement being able to communicate hydrophobia to a healthy animal when he says: "The hypothesis that certain ferments—an improper term—may be developed in great abundance in the saliva under the influence of psychical disturbance, would account for those instances in which rabies shows itself in dogs bitten by others which are excited or furious by sexual desire, though themselves healthy."

It would appear that the saliva is the only means of communication. Dupuytren, Breschet, and Magendie, endeavored to convey the disease by injecting the blood of dogs, suffering from rabies, into the veins of healthy dogs, but always unsuccessfully. The flesh, milk, semen, and abdominal secretions, were likewise found not to be media for transmission.

On the other hand, Eckel, of Vienna, after several failures, inoculated a dog with the blood of a man who was affected with hydrophobia. On the sixty-second day thereafter the animal was seized with unmistakable rabies and died. Fleming, however, from whom I quote this statement, says that it must not be forgotten that, at the time of these experiments, rabies was raging as an epizoötic. But Bouley,[2] who has investigated the whole subject of hydrophobia with great ability, declares that it can be transmitted only by inoculation, and the only agent which has the power of communicating it is the saliva, in which alone the virus exists. Any other liquid taken from a rabid animal is ineffective. Inoculation by blood, even its transfusion, has failed to produce any results. He also says that all living beings affected with hydrophobia are capable of transmitting it ; that is, the saliva of all rabid animals is virulent, it matters not to what species they belong.

Whether or not it originates spontaneously in the lower animals, it is very certain that it has no other origin in man than inoculation.

Although there is no sure evidence on the point, there appears to be no room to doubt that hydrophobia may be communicated by inoculation from a person affected with the disease to an unaffected individual. Aurelianus, Enaux, and Chaussier, and others cited by Fleming, mention instances in which it has been induced in persons who have accidentally had the saliva of hydrophobic patients applied to their lips. Fleming[3] states that in 1871 a girl named Bence died in Liverpool from hydrophobia. It was believed she had not been bitten, but the death of her little brother, from the disease, occurred about three weeks previously, and the supposition was that the virus had been communicated in some way to the girl through a wound in her foot.

---

[1] "Rabies and Hydrophobia," London, 1872, p. 124.

[2] "Hydrophobia," by H. Bouley, translated from the French by A. Liautaud, M. D., V. S., New York, 1874, p. 6.

[3] *Op. cit.*, p. 141.

The fact that hydrophobia can be communicated from man to the lower animals is sufficiently well established by the experiments of Magendie, Breschet, Earle, and Renault.

The wolf is said to be the most dangerous of all animals when rabid, for the reason probably that it seizes the neck or face, parts not fully protected by clothing, and thus the saliva is not so apt to be rubbed off as when the leg, for instance, is the part attacked.

The slightest abrasion of the skin coming in contact with the saliva may be sufficient for inoculation. Cases are recorded in which the disease has resulted from dogs licking the hand or face on which there were pimples or sores.

Diagnosis.—That protean disease, hysteria, occasionally puts on the semblance of hydrophobia. Several cases of the kind have occurred to me, and, in all, the symptoms were in general character very much like those which are exhibited by genuine hydrophobia, though in some respects, perhaps, a little exaggerated. It will in these and similar cases —the result of fright and imagination—often be found that the patient has been bitten by a dog not long before. There is a want of consistency about the symptoms which of itself is sufficient to excite suspicion as to the real character of the phenomena. Thus, although at times the attempt to swallow will excite laryngeal and other spasms, these do not always occur under similar circumstances, and are not induced by those secondary and more refined influences, such as the sound of falling water, bright lights in the face, excitations applied to the skin, seeing others drink, etc., which so generally cause them in the real disease. There are not the same anxiety and depression in the simulated disease as in the real, though the apparent emotional disturbance is much greater. The hysterical patient is loud in the expression of apprehensions, while the real hydrophobic one, though intensely anxious and terrified, endeavors to prevent others perceiving the state of his mind.

The history of the case, the existence of the hysterical diathesis, and the fact that the symptoms come on soon after the bite without any period of incubation, will further aid in establishing the diagnosis between the false and the real disease.

The last case of the simulated disease which has come under my observation was that of a policeman whom I saw in consultation with Dr. S. G. Cook in the summer of 1874. The man was then in the Park Hospital, held down on a bed, and snapping like a dog at every person who came in his way. At the sight of water he became intensely excited, foamed at the mouth, and went through a series of fearful contortions of his limbs. But, when I took a glass of water in my hand and told him in a commanding voice to drink immediately, he swallowed the liquid without the slightest difficulty.

The bromide of potassium in large doses was prescribed, and the next day all his symptoms had disappeared. On inquiry it was ascertained that he had been bitten by a dog several days before, and that his comrades had frightened him by their inquiries and suggestions.

The fact that a disease resembling hydrophobia may be induced by physical derangement and by mental disturbance especially of the imagination, and that death may be the consequence, is very well established, and may account for the apparently spontaneous instances, and for those cases of long incubation which are cited by authors.

Thus M. Labadie Lagrave[1] quotes from Raymond (de Marseille) the case of a child twelve years old who became hydrophobic without known cause and died at the end of ten days. Also a case from Rouppe of a sailor who had convulsions and died hydrophobic without known cause, and another from Pouteau of a man who died in fifteen hours with symptoms of hydrophobia which had ensued on a violent paroxysm of anger.

Berthier[2] refers to several similar cases occurring as the result of menstrual derangement.

Fleming[3] cites the instance of a woman who had been bitten in the face and who was admitted to the Hôtel-Dieu in Paris. After a few days she was cured of her wounds and discharged. Going about her usual avocations one day she heard a man exclaim, "She has not gone mad, then!" From that time she could not swallow liquids, and on the same day was readmitted to the Hôtel-Dieu, and this time to die of hydrophobia.

The following case is also given by Fleming: "A woman in the clinic of Dr. Maisonneuve had been bitten by a dog, which was supposed not to be rabid, and the injury had healed; when two months after the accident she was met by two students, who had been with the doctor at the time, and who asked her if she was not yet mad. Immediately she was seized with nervous symptoms, became intensely anxious and uneasy, and went into the hospital in the belief that she was hydrophobic. She was put under the care of M. Laugier and the following day was evidently affected with the disease; hemiplegia appeared, with a violent delirium, accompanied by an irrepressible amount of fear, and she died asphyxiated in forty-eight hours."

The temperature in all cases of pseudo-hydrophobia that I have witnessed was not above the normal standard.

Hydrophobia has been confounded with tetanus, and some writers have regarded it as a modified form of this affection. The distinction is, however, so well marked that it scarcely seems necessary to dwell upon it. The facts that in tetanus the spasms are tonic, while in hydrophobia they are clonic; that in the first-named they are mainly shown as regards the jaws and back, while in the latter they radiate from the throat; that in tetanus the mind is clear throughout, while in hydrophobia more or less mental implication is always present, will suffice to render any mistake in the diagnosis of the two diseases impossible.

---

[1] Article "Hydrophobie," in "Nouveau dictionnaire de médecine et de chirurgie pratiques," tome xviii., Paris, 1874, p. 17.

[2] "Des névroses menstruelles," Paris, 1874, p. 169.

[3] *Op. cit.*, p. 176.

From epilepsy the distinction is so obvious as not to require further mention.

Prognosis.—There is no authentic instance on record of a cure of hydrophobia. Several such have been reported, but inquiry has always shown misstatement or error somewhere. The fact that the hysterical counterpart has several times been regarded as the real disease, is the main support for the opinion of some authors that the affection is curable.

Several years ago Dr. Ligget,[1] of Maryland, reported a case of hydrophobia cured by calomel. A careful examination of the details of this case excites very grave doubts in my mind in regard to its really being an instance of the disease in question.

The subject was a negro-woman who had been bitten about two weeks before any symptoms were manifested. The dog was lying quietly in the yard, and bit her in the great-toe as she was teasing him with her foot. The animal was at once chained up, and died in two or three days with "all the symptoms of *rabies canina* in its most virulent form." It does not appear that the doctor saw the dog, and it is very probable that the rigid confinement would have caused the animal to exhibit symptoms which would easily be mistaken by laymen for those of hydrophobia.

Again, the period of incubation was unusually short, and the symptoms, as detailed by Dr. Ligget, are clearly not those of hydrophobia. Thus, although he repeatedly states that there was inability to swallow liquids, there is no distinct mention made of the pathognomonic laryngeal and pharyngeal spasms which occur in hydrophobia, and which are so frightful in character. The convulsions all appear to have been general, and there was a "horror" of water, which is not a phenomenon of the true disease. For these reasons I am constrained to believe that the disease treated by drachm-doses of calomel was in reality one of hysteria which assumed the form of hydrophobia. In this opinion I am sustained by an eminent medical gentleman residing in Dr. Ligget's neighborhood, who, as the latter admits, declared the affection to be "a case of that protean disease, hysteria, simulating hydrophobia." Calomel has been repeatedly tried before and since Dr. Ligget's case, but without effect.

---

[1] "Case of Hydrophobia successfully treated with Drachm-Doses of Calomel," *American Journal of Medical Science*, January, 1860, p. 96.

But, although the prognosis is so hopeless in the developed disease, it is much more favorable as regards the probability of the supervention of hydrophobia from the bites of rabid animals, for, of those bitten by dogs unmistakably affected with the disease, not more than one in fifteen becomes successfully inoculated. This liability differs greatly according to the circumstance of the part being covered or not. The wounds of the face, neck, or hands, are much more likely to be followed by hydrophobia than those inflicted on the legs or feet, where the virus is rubbed off by the clothing before the teeth reach the flesh. The bite of a rabid wolf is more apt to be followed by the disease than the bite of a dog, for the reason that the first-named generally seizes the throat or face. Thus, Trolliet states that at Brives, in France, seventeen persons were bitten by a rabid wolf, of whom ten died of hydrophobia; and, of twenty-three bitten by another, thirteen died. On the other hand, Hunter states that on one occasion a dog bit twenty persons, of whom only one was inoculated. Those first bitten by a rabid animal are more liable to have hydrophobia than those bitten subsequently, when the poison is in a measure exhausted. Probably the most dangerous wounds are those which barely penetrate the epidermis, and in which, therefore, the venom is not washed away by any flow of blood.

Morbid Anatomy.—Within the last few years the study of the morbid anatomy of hydrophobia has led to results which may be considered, at least for the present, as determining, with some degree of exactness, the situation and character of the essential lesions of this terrible disease.

In 1869 Meynert examined microscopically the spinal cords of a boy and girl, patients at Oppolzer's clinic, who died of hydrophobia.

In the first case, he found thickening of the walls of the spinal vessels, amyloid degeneration and nuclear proliferation of the cells of the neuroglia.

In the second case, the neuroglia of the posterior columns of the cord was hypertrophied, through swelling of the stellate bodies. In the antero-lateral columns there were granular and amyloid degeneration, and numerous distended blood-vessels.

The cortical substance of the brain exhibited the presence of a large number of lacunæ with colloid masses. The nerve-cells of this part were the seat partly of molecular disintegration, and partly of sclerotic enlargement.

Next are the observations of Dr. Clifford Allbutt,[1] who examined the nerve-centres in two patients, who died of hydrophobia while inmates of the Leeds General Infirmary. Throughout the brain and spinal cord there were evidences of great vascular congestion with transudation into the surrounding tissue. In several places the walls of the vessels were thickened and there were here and there patches of incipient nuclear proliferation. There were also hæmorrhages into the medulla oblongata. In many places there was a refracting material to be seen outside of the vessels, which probably was of the nature of a coagulated fibrinous exudation. Finally, Dr. Allbutt found in the encephalon occasionally, and in both spinal cords, and especially in both medullas, little gaps caused by the disappearance of nerve-strands which had passed through the granular degeneration of Clarke. These phenomena, adds Dr. Allbutt, point to the action of an animal poison acting primarily on the cerebro-spinal nervous system.

---

[1] "Specimens illustrating the Pathological Anatomy of Hydrophobia," "Transactions of the Pathological Society of London," vol. xxiii., p. 16, 1872.

Then in July, 1874, were my own researches,[1] made at Dr. Hadden's request, in the case, the details of which, as observed during life, have just been given.

As preliminary to the description of the microscopical appearances, it may be stated that, on removing the calvarium, the membranes of the brain were found to be congested, but there was no appearance of serous effusion to an abnormal extent either in the sub-arachnoidal space or in the ventricles. The substance of the brain was only slightly congested, but the consistence, especially of the cortical tissue, was somewhat less than normal. The cerebellum appeared to be healthy, as did also the pons Varolii, the corpus striatum, the optic thalamus, and other ganglia, with the exception of the medulla oblongata, which seemed to be slightly softened. The membranes covering it and the upper part of the spinal cord were congested.

I took for examination (1) portions of the cortical substance of the brain; (2) sections of the corpus striatum; (3) sections of the optic thalamus; (4) sections of the cerebellum; (5) the pons Varolii; (6) the medulla oblongata; (7) a section of the spinal cord at the level of the second pair of cervical nerves; (8) a portion of the pneumogastric nerve from the neck:

1. Cortical substance of the brain.

My examinations of this tissue were made upon specimens which had been kept in absolute alcohol eighteen hours, in glass tubes surrounded with ice. I experienced no difficulty in cutting sufficiently thin sections. In all the sections the following conditions existed (object-glass one-fourth inch):

a. The blood-vessels were increased in size and number, and their walls appeared to be thickened.

b. There were minute extravasations of blood throughout, in some of which the blood-disks could still be distinguished, but in most of them they were broken down.

c. The external layer of nerve-cells had almost entirely been replaced by fatty matter in the form of oil-globules. The cells that remained were filled with a highly-refracting granular material, which was also oil in very minute particles. None of these cells were bi-nuclear. Amyloid corpuscles were discovered generally at the junction of this with the next stratum.

---

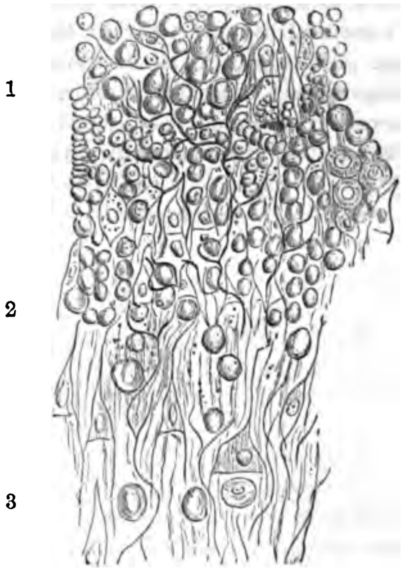
[1] "Proceedings of the New York Neurological Society," July 7, 1874, in *Psychological and Medico-Legal Journal*, September, 1874, p. 169.

d. The second layer of cells had also to a great degree been replaced by fat, but not to the same extent as the outer layer. It is well known that this layer is composed of more numerous and larger cells than the outer; but there was no doubt of their atrophy or disappearance.

e. The third layer, composed of large cells, was scarcely affected. A few oil-globules were seen, and occasionally an amyloid corpuscle. The remaining strata were not involved, so far as I could see, to the slighest extent.

In Fig. 94 a vertical section of the cortical substance is seen: 1, the outer or peripheral stratum; 2, the second layer; 3, the third layer or large cells.

FIG. 94.



2. The corpus striatum, the optic thalamus, and the cerebellum were in an apparently normal condition, though there was some evidence of arterial injection.

3. The pons Varolii was not examined in the fresh state, but was placed entire in a solution of bichromate of potash to harden. Subsequently examined, it was found to be the seat of extravasation of blood, and the vessels were enlarged and their walls thickened.

4. The greater portion of the medulla oblongata was also placed in

the bichromate of potash solution, but several sections were made after the part had been in absolute alcohol surrounded by ice for twenty-four hours.

a. The first of these was made through the olivary bodies, at the level of the floor of the fourth ventricle, so as to include the nuclei of the pneumogastric and hypoglossal nerves.

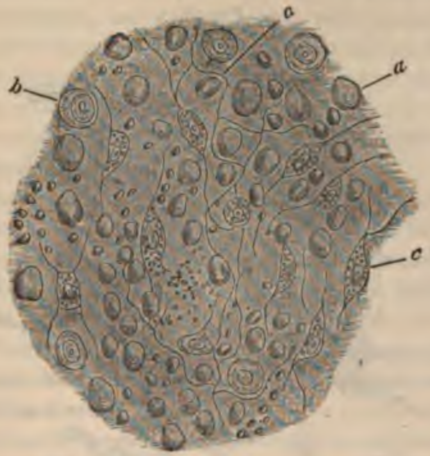
Numerous extravasations of blood could be seen with the naked eye, but with an inch objective they were more clearly made out. The vessels were then seen to be enlarged and more numerous than in the normal condition. The gray matter forming the nuclei of the pneumogastric and hypoglossal nerves was observed to be of a distinctly granular appearance, and the roots of the nerves presented a like characteristic. In other respects the section exhibited nothing abnormal.

b. Examined with a fourth-inch objective, this granular matter of the nuclei was seen to consist of oil-globules and amyloid corpuscles. The cells were ascertained to be atrophied both in size and numbers. Indeed, they had almost entirely disappeared. Of course it was not possible, in a fresh and unprepared preparation, to form any definite idea of the relative proportion of nerve to neuroglia cells, but the deficiency of all cell-structure was very remarkable.

(Fig. 95, *a*, oil-globules; *b*, amyloid bodies; *c*, nerve-cells; *d*, blood-vessels.)

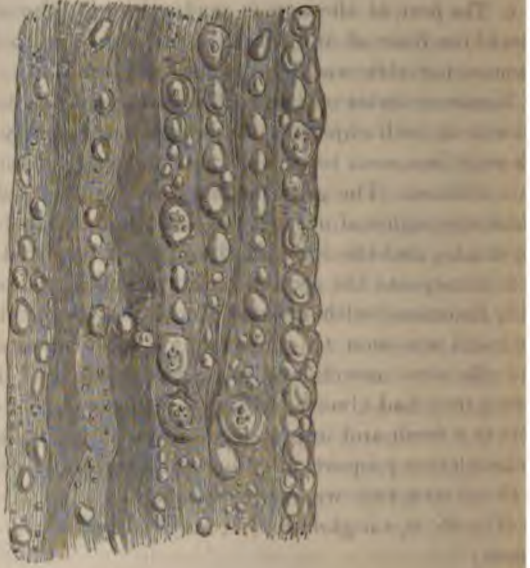
c. The nerve-roots, when examined in like manner, were seen to have undergone a similar change, the granular matter consisting entirely of fat, mainly in the form of oil-globules (Fig. 96).

FIG. 95.



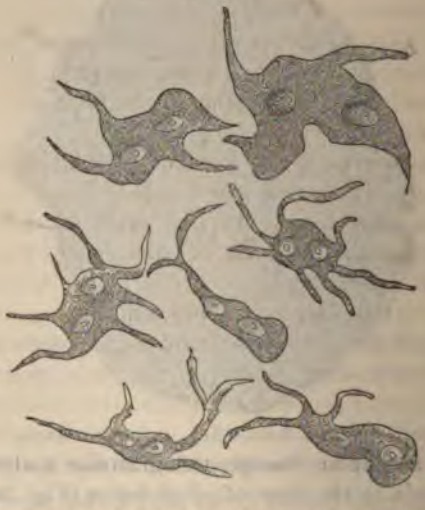
Sections made immediately below the level of the point of the calamus scriptorius, so as to include the main root of the spinal accessory nerve and its nucleus, exhibited almost exactly the same appea ances.

Fig. 96.



5. *The Spinal Cord.*—The section of the cord was made at a poi about midway between the first and second cervical nerves. The gr

Fig. 97.



matter of the anterior and posterior horns was found in a state of granu- lar and fatty degeneration, the cells atrophied, and the nerve-roots in a similar condition. In the white matter, both of the anterior and posterior columns, there was nuclear proliferation of the neuroglia-cells (Fig. 97).

6. The peripheral portion of the pneumogastric nerve, carefully removed by my assistant and placed in strong alcohol, exhibited a red appearance, but this may have been due to imbibition.

Benedict,[1] about the time of my own observations, made a series of researches into the morbid anatomy of hydrophobia as met with in dogs. His results were—

1. The vessels situated between the cerebral convolutions were distended with blood, and their external walls were coated with an exudation of a highly-refractive material consisting of granules.

2. Numerous cavities were found to exist in the gray matter of the brain, and these were filled with a like granular, highly-refracting material similar to that found in the walls of the vessels.

3. Masses of myeline, indicative of softening, and chemical changes of the nerve-tissue, were also discovered.

Benedict regards the appearances as identical with those which Lockhart Clarke has considered as indicating granular degeneration.

From the foregoing data it will be perceived that at last something definite has been ascertained relative to the morbid anatomy of hydrophobia. Whether we regard the condition, according to Benedict, as an acute exudative inflammation, or as a granular degeneration, is of no consequence so far as the facts are concerned. Whether on the one hand the granular matter is an exudation, or whether it results from degeneration of the nerve-tissue, are points which will probably ere long be cleared up. My own view is in accordance with that of Lockhart Clarke, who, detecting a like change in other affections of the nerve-centres, views it not as an exudation but as a degeneration.

As to the gross lesions, congestion of the brain and spinal cord has been found by many observers.

Sometimes the nerves at the wound are inflamed, but this is not a uniform occurrence. The eighth pair has been found to present a pinkish appearance in some cases. In four cases in which the blood was examined by Schivardi,[2] infusoria of the genera *bacterium*, *monas*, *vibrio*, and *torula*, existed.

The fauces, pharynx, larynx, trachea, and lungs, are generally found reddened and congested, as much from the asphyxia as from any specific influence of the disease.

Pathology.—Even if we had no information relative to the morbid anatomy of hydrophobia, no one who has ever witnessed a case could fail to perceive the implication of the hemispheres, the medulla oblongata, and the spinal cord. The hallucinations and other mental phenomena point to the hemispheres, the irregular action of the respiratory muscles, and the heart, together with the gastric derangement and pharyngeal convulsions, indicates the implication of the pneumogastric nerves, and the spasms of the larynx point to the origins of the spinal accessory nerves in the spinal cord. Since we have arrived at some degree of exactness relative to the lesions in the disease, we cannot fail to have our conviction on these points strengthened.

---

[1] "Die anatomischen Veränderungen bei der Lyssa des Hundes," *Wiener Medizinische Presse*, July 5, 1874.

[2] "Observations nouvelles sur la rage," Besançon, 1868, p. 22.

The nature of the virus is unknown. It is probably of the nature of a ferment, but this cannot be regarded as satisfactorily proved.

In 1820, Dr. Marochetti observed, in the Ukraine, that during the formative period of hydrophobia small vesicles or pustules formed under the tongue, and that, if these were opened and cauterized, the further development of the disease was prevented. I have never been able to find these formations, but they were recognized, two years after Marochetti published his account, by Magistral, in France. This latter opened and cauterized them in the manner recommended by Marochetti in ten cases, in five of which, nevertheless, the affection went on to full development, and the patients died. I am not aware that any one else has discovered these pustules.

For full details relative to hydrophobia as it appears in dogs, I must refer the reader to the late Mr. Youatt's excellent book on canine madness, and to the more recent and thorough treatise of Fleming. I may, however, state that it is very clearly established that canine rabies is not so frequent in very hot as it is in temperate or cold weather; that it is not induced by thirst or improper food, or by preventing copulation.

Is hydrophobia primarily a disease of the nerve-centres or a blood-disease? I suppose it is utterly impossible, in the present state of our knowledge, to answer such a question. It may start as a blood-disease and end as a nerve-disease. Blood-diseases lead to structural changes of various organs of the body, and the nerve-centres are likewise involved to a considerable extent. Is it not worth while to call attention to the numerous instances of blood-diseases which produce structural changes? Hydrophobia may be a blood-disease, and yet afterward be succeeded by changes in the nerve-centres. It is not necessary to suppose that hydrophobia is a nerve-disease from the beginning. It is perfectly possible, however, that it may be, and there are a great many instances which can readily be adduced in proof of this assertion. Take tetanus for example. Very few pathologists pretend to say that tetanus is a blood-disease. It is a disease propagated through the nerve-tissue starting from injury of a peripheral nerve, and inducing structural changes in the spinal cord. Dr. Lockhart Clarke, as we have seen, has ascertained in a number of cases that the essential condition of tetanus is a granular degeneration of the cord, and that is, probably, only the beginning of the fatty degeneration I find in hydrophobia, and yet there is no suspicion of blood-poisoning in tetanus. Hydrophobia presents many analogies to tetanus, not only in its morbid anatomy but in its natural history.

Epilepsy can be caused by injuries to peripheral nerves. I had a case some years ago of a lady who wounded her thumb, and six months afterward she had epileptic paroxysms, which were preceded by an aura originating in the cicatrix. And if epilepsy—which is another one of the spasmodic diseases—can be induced by a simple wound, why not hydrophobia? So that we have examples of analogous diseases caused by wounds of nerves, without the necessity of supposing the blood to be primarily affected.

Still, there cannot be much doubt that the poison in the saliva, and not the wound made by the animal's teeth, is the essential influence producing hydrophobia. It is not at all certain, however, that the latter may not in some cases produce a modification of the characteristics of the disease, perhaps causing those tetanoid phenomena which are occasionally present.

Treatment.—The measures of treatment relate to those proper immediately after the infliction of the wound, with the view of preventing the development of the disease, and those advisable after the affection is unmistakably manifested.

Under the first category comes excision, which should be performed as soon as possible, and which is probably the best of all prophylactics. The operation should not be done with a niggardly hand, but every part with which the teeth of the animal have come in contact should be removed, as well as the tissue into which the poison may have become infiltrated. Previous to the operation, in fact as soon as the wound has been received, a tight ligature should be bound around the limb immediately above the injury, and, after the knife has done its work, cupping-glasses should be applied over the spot, till the tissues in the vicinity are thoroughly drained of blood. I have performed excision, for the wounds received from dogs certainly rabid, eleven times, and always with the effect of preventing hydrophobia.

Cauterization may be performed instead of excision, and is preferred by some practitioners. Mr. Youatt used it with over four hundred persons bitten by rabid animals, and never unsuccessfully. Four times he employed it on himself, but there is a strong probability that the practice at last failed with Mr. Youatt himself, for he committed suicide while supposed to be suffering from the initial symptoms of hydrophobia.

He preferred the nitrate of silver as an escharotic. Others have made use of the actual cautery, caustic alkalies, the mineral acids, arsenic, chloride of zinc, and carbolic acid. I have employed cauterization seven times—four with the nitrate of silver and three with the actual cautery—upon persons bitten by rabid dogs, and always with success.

Mr. Youatt at one time had faith that the scutellaria lateriflora, or scullcap, was a preventive. He moistened three pieces of tape with the saliva of a rabid dog, and inserted them as rowels into the skin of three dogs. To two of these he gave scutellaria combined with belladonna, while the third was left to itself. On the twenty-ninth day after the inoculation this latter became rabid, while the others, several months afterward, were alive and well.

Notwithstanding this experience, it would not be justifiable in the physician to neglect performing either excision or cauterization as soon as possible after the reception of the bite. Even if several weeks or months have elapsed, one or the other—preferably excision—should be performed.

As to the treatment of the fully-developed disease, there is nothing, in my opinion, which has hitherto succeeded in arresting its onward course. Cases of cure have been reported, but, as already stated, they are open to the suspicion of not being true instances of the disease. Excessive bloodletting has been reported as a successful remedy; injection of warm water into the veins dissipated the paroxysms in a case reported by Magendie, the patient, however, dying; and nearly every stimulant, narcotic and sedative, in the materia medica, has been used. In the case which I saw with Dr. Cook, and which has already been cited, the hydrate of chloral was administered. The effect certainly was to mitigate the severity and frequency of the spasms, but it was, as Dr. Cook states, given too late in the course of the disease to produce any permanently curative result. In the present state of our knowledge I should be more disposed to rely on the hot-air bath at a temperature of about 200° Fahr., and the administration of hydrate of chloral in large doses frequently repeated, than on any other plan of treatment. In Dr. Cook's case the Turkish bath was proposed, but the parents of the child would not consent to its use. Hypodermic injections of morphia and atropia may be used with some advantage to mitigate the force of the paroxysms.

Before concluding my remarks on hydrophobia, it is proper to allude to the attempts of Dr. Schivardi,[1] of Milan, to cure the disease by the primary galvanic current. In one case the current was feeble, and was continued for nineteen hours. Great improvement ensued; the oppression disappeared, and the dysphagia was entirely relieved. Through some misunderstanding, advantage was not taken of these ameliorations, and the patient was allowed to die.

In the other case, which was one of undoubted hydrophobia, occurring in a girl nine years old, the current from twenty-two Daniell's cells was employed. The current was passed from the soles of the feet to the forehead for fifty-eight hours almost continuously, and the duration of the disease prolonged to seven days and seven hours, when the patient died. During the last two days there were no hydrophobic symptoms.

---

[1] "Observations nouvelles sur la rage."

Further trials are necessary before the therapeutical value of galvanism in hydrophobia can be ascertained.

CHAPTER II.

EPILEPSY.

Epilepsy, although only a symptom of a morbid condition, must for the present be considered as a disease, for the reason that we are not able to designate with certainty its exact seat, or the nature of the lesion which exists. It is characterized by paroxysms of more or less frequency and severity, during which consciousness is lost, and which may or may not be marked by slight spasm, or partial or general convulsions, or mental aberration, or by all of these circumstances collectively. The essential element of the epileptic paroxysm is loss of consciousness. Without that there is no true, fully-formed epileptic paroxysm.

Symptoms.—Although in many cases there are no precursory phenomena, it often happens that there are indications of an approaching attack. These are exceedingly variable in character and situation. They may consist of pain in the head, a sensation of constriction or fullness, vertigo, noises in the ears, a feeling as if the ears are stopped with cotton or water, flashes of light, or sudden blindness, illusions or hallucinations of any of the senses—irritability of temper, extraordinary cheerfulness, difficulties of speech, pains in various parts of the body, especially in the stomach, bowels, or ovaries, sensations of numbness or of tingling, or of an indescribable character, which begin in an extremity or in some other region, and appear to pass rapidly to the head —a feeling of constriction in the throat, vomiting, sudden evacuation of the bladder or rectum, erections of the penis, with or without the sexual orgasm, and discharge of semen, with many others of almost every possible description.

The prodromata may precede the attack by a considerable period, but usually are only a few moments in advance of it. Indeed, often the interval is so short that they may be regarded as a part of the paroxysm.

The sensations of numbness or tingling, or of an electric shock, as a sharp stab, or blow, or pain, which precede the attack and which originate in different parts of the body, and in some cases seem to run rapidly toward the head, are called auræ. Sometimes this aura is fixed, and may consist of various derangements of sensation besides those above mentioned. In a number of my patients it has been a sensation at the pit of the stomach, such as that produced by a slight feeling of hunger or of anxiety. Again, it has consisted of a sharp impression on the tongue; at others of a subjective sense of smell, and again colored visions, or hallucinations of sight.

In regard to these auræ of colors, Dr. Hughlings Jackson[1] has made some interesting observations. He finds that red is the color which is usually seen first, though the others may follow in such rapid succession as to present an image of all the primary colors. Loss of the power to see colors (color-blindness) is generally first shown as regards red; and if this affection advances, the insensibility is progressively shown toward the violet end of the spectrum. So in the epileptic chromatic hyperæsthesia, the formation of colors is in the same direction, and hence red is first perceived and violet last—theoretically, at least, for there are not yet sufficient data collected to enable us to speak with any degree of certainty on the subject. There are exceptions, however, for Dr. Jackson cites the case of one of his patients who always saw blue just before an attack. In my own experience, red has been invariably the predominating color, and in most cases the only one. The case of the gentleman who, just before his paroxysm of epilepsy, saw an old woman clothed in red approach him, with a stick raised in a threatening manner, and the fit coming on as soon as the blow fell on his head, is well known. Two similar instances have come under my own notice.

Other derangements of sight may coexist with the chromatism as epileptic auræ. Thus, Sauvages[2] mentions the fact that a woman subject to epilepsy saw during the paroxysm dreadful spectres, and that real objects appeared magnified to an extraordinary degree; a fly seemed as large as a fowl, and a fowl appeared equal in size to an ox. In colored objects, green predominated with her, a fact which Ferrier states he has met with in other convulsive diseases. He also states that a very intelligent boy, who was under his care for convulsions of the voluntary muscles, when he looked at some large caricatures, glaringly colored with red and yellow, insisted on it that they were covered with green, till his paroxysm abated, "during which his intellects had not been at all affected."

A young lady, who had overtasked her mind at school, was thrown thereby into what I regarded as a more or less hysterical condition, but which some authorities would probably consider epileptic. She saw spectres of various kinds all day, but every real object at which she looked appeared to be of an enormous size: a head, for instance, seemed to be several feet in diameter, and little children looked like giants.

---

[1] *British Medical Journal*, February 7, 1874.

[2] Reported by Ferrier, in "An Essay toward a Theory of Apparitions," London, 1813 p 86.

When I took out my watch, while examining her pulse, she remarked that it was as large as the wheel of a carriage.

In the case of a young gentleman, now under my care for epilepsy, the attacks are invariably preceded by a period which lasts several hours and sometimes a whole day, during which he "sees small." Every thing appears to be of infinitesimal size. This phenomenon I have never seen noted by any other writer on epilepsy.

Auræ connected with the sense of hearing are uncommon, except such as merely consist of tinnitus—roaring, buzzing, singing, etc.—these are often met with. But in one case there were distinct hallucinations of hearing preceding the attack, the patient always fancying that he heard his name repeatedly called.

An aura may be entirely manifested by dreams or delusions. As an instance of the first I quote the following remarkable case from my treatise on "Sleep and its Derangements." The patient occasionally visits me for medical advice, but has had no epileptic paroxysm for over four years.

"A lady of decided good sense had an epileptic seizure, which was preceded by a singular dream. She had gone to bed feeling somewhat fatigued with the labors of the day, which had consisted in attending three or four morning receptions, winding up with a dinner-party. She had scarcely fallen asleep when she dreamed that an old man clothed in black approached her, holding an iron crown of great weight in his hands. As he came nearer she perceived that it was her father, who had been dead several years, but whose features she distinctly recollected. Holding the crown at arm's length, he said: 'My daughter, during my lifetime I was forced to wear this crown; death relieved me of the burden, but it now descends to you.' Saying which, he placed the crown on her head and disappeared gradually from her sight. Immediately she felt a great weight and an intense feeling of constriction in her head. To add to her distress she imagined that the rim of the crown was studded on the inside with sharp points which wounded her forehead so that the blood streamed down her face. She awoke with agitation, excited, but felt nothing uncomfortable. Looking at the clock on the mantelpiece, she found that she had been in bed exactly thirty-five minutes. She returned to bed and soon fell asleep, but was again awakened by a similar dream. This time the apparition reproached her for not being willing to wear the crown. She had been in bed this last time over three hours before awakening. Again she fell asleep, and again, at broad daylight, was awakened by a like dream.

"She now got up, took a bath, and proceeded to dress herself, with her maid's assistance. Recalling the particulars of her dream, she recollected that she had heard her father say one day that in his youth, while in England, his native country, he had been subject to epileptic convulsions, consequent on a fall from a tree, and that he had been cured by having the operation of trephining performed by a distinguished London surgeon.

"Though by no means superstitious, the dreams made a deep impression upon her, and, her sister entering the room at the time, she proceeded to detail them to her. While thus engaged she suddenly gave a loud scream, became unconscious, and fell upon the floor, in a true epileptic convulsion. This paroxysm was not a very severe one. It was followed in about a week by another, and, strange to say, this was preceded as the first by a dream of her father placing an iron crown on her head, and of pain being thereby produced."

Subsequently this lady had two other attacks, at intervals of several months, and both were preceded by the dream of the iron crown.

In the case of a gentleman formerly under my treatment for epilepsy, the fits were invariably preceded by dreams of troubles of the head, such as decapitation, hanging, perforation with an auger, etc.

It is probable that in such cases as the foregoing, the dream is excited, as dreams often are, by derangements of sensibility, which are themselves the auræ.

In some cases the auræ are entirely psychical, consisting of illusions, hallucinations, or delusions. Delusions are not common as auræ. I have, however, had one case in a lady, who had an epileptic seizure immediately after hearing of the death of a gentleman to whom she was engaged to be married, and whose subsequent paroxysms were almost always preceded by the delusion that she was going to be killed. There was no exaggeration of motility, but the delusion was firmly held and acted upon, to the extent that she would give away her effects, and make other preparations for her death. The following day the fit usually occurred, although sometimes it was delayed for two days.

Delasiauve,[1] of two hundred and sixty-four cases, found the paroxysms unannounced in one hundred and one, and with precursory phenomena in one hundred and eighty-three. The prodromata were immediate in one hundred and fifty cases. These he divides into seven categories, as follows. It is to be recollected that cases may appear under one or more categories, according as the prodromata, as is often the case, are met with simultaneously in different parts of the body:

FIRST SERIES.—*Precursory Signs in the Head.*—Seventy-five cases.

Vertigo, flashes of light	33
Headache, weight in the head	15
Heat of face	3
Various localized sensations	13
Indefinite sensations	1
Illusions, hallucinations, and other sensorial aberrations	9
Rotation of the head or of the eyes	5
Grinding of the teeth, derangement of the motility of the tongue	2
Tendency to sleep	1
Constriction of the throat	3

---

[1] "Traité de l'épilepsie—histoire—traitement—médecine légale," Paris, 1854, p. 47.

Second Series.—*Precursory Signs in the Throat.*—Twenty-two cases.

Oppression of the chest and sense of suffocation	9
Sensation of a ball or of motion in the pectoral region	2
Shivering sensation of cold or of an aura	5
Pain or heat	4
Palpitations, spasms	2

Third Series.—*Precursory Signs in the Abdomen.*—Thirty-two cases.

Pain with or without oppression, eructations, vomiting	13
Intestinal or uterine colic	3
Sensation of a ball	3
Sensation of cold, of a vapor, etc.	6
Stomachal heat	1
Undefinable sensations	6

Fourth Series.—*Precursory Signs in the Extremities.*—Ninety four cases.

Numbness, contractions, jerkings, retractions, cramps, formications, etc.	36
Pain with or without spasms	13
Tremblings	10
Aura or phenomena approaching thereto	20
Undefinable sensations	15

Fifth Series.—*Precursory Signs, consisting of General and Undefinable Sensations.*—Twenty-two cases.

General agitation or rotation of the body	8
Condition of discomfort, fainting, etc.	6
Vague sensations	7
Moroseness	1

Sixth Series.—*Precursory Signs situated in the Genital Organs.*—Five cases, such as retraction of the testicles, aura starting from the testicles and spermatic cords, sensations located in the uterus, etc.

Seventh Series.—*Exceptional Cases.*—Desire to defecate, to urinate, profuse perspiration, etc.

The Paroxysm.—Great differences are observed in the character and severity of the paroxysm. Ordinarily two varieties are recognized, the *petit mal* or slight attack, and the *grand mal* or severe seizure. The first is unattended by marked spasm or agitation; the latter is characterized by more or less violent tonic and clonic convulsions. These divisions are, however, not regarded as sufficiently precise by those who have studied the disease in question with care and precision, and more minute classifications of the phenomena of the epileptic paroxysm have accordingly been made. The one which I have used in my lectures at the University Medical College for several years past is less complex than some others, and embraces all the known varieties. It is as follows:

1. Momentary unconsciousness without marked spasm.
2. Unconsciousness with evident though local spasm.
3. Unconsciousness with general tonic and clonic convulsions.
4. Irregular or aborted paroxysms.
5. Recent investigations have led me to the recognition of a distinct form of epilepsy characterized by hallucinations, and to which I have ventured to propose the name Thalamic Epilepsy.

Besides these several varieties, there are certain accompaniments, such as hysteria, mania, and paralysis, which will require consideration.

1. *Momentary Unconsciousness without Evident Spasm.*—The patient is perhaps standing, engaged in conversation, when a momentary blank in his mental processes occurs. It probably does not attract attention; it is instantaneous, disappears, leaving no feeling of discomfort after it, and there is an almost immediate continuance of his thoughts and speech. Or he may be walking in the street when the accession occurs. He loses himself for an instant, but he continues to walk, and does not even stagger.

In somewhat more severe seizures, if conversing, he stops suddenly, stares vacantly but fixedly for a moment, and may drop anything which he has in his hand.

If walking, his steps are arrested for an instant, he staggers, and would fall but for the quick return of consciousness.

Such is the general character of these absences, faints, spells, etc., as they are popularly called; varying, however, according to the circumstances of the moment and the condition of the patient. They frequently exist for a long time without the patient paying much attention to them. In a gentleman now under my charge they occurred several times in the course of the day when walking, riding on horseback, sitting quietly in his library, engaged in conversation, or eating. The continuity of his acts was scarcely interrupted, and those about him never noticed that anything was wrong.

In the case of a young lady they occur generally at the dinner-table. She drops her knife and fork, looks steadily to the front, ceases to eat, and in about two seconds resumes her occupation with a long-drawn inspiration. Those near her observe that her countenance becomes very pale, and that she does not hear or see.

Sometimes these attacks, slight as they are, are followed by pain in the head, vertigo, confusion of ideas, numbness, and other evidences of nervous derangement, which may last for several hours, and which become more pronounced as the epileptic condition becomes more confirmed.

2. *Unconsciousness, with Evident though Local Spasm.*—In this variety the loss of consciousness is of longer duration than in the pre-

ceding, and is attended with convulsions light in character, but yet apparent to those around. The eyes are fixed, as in the first variety, the mind becomes a blank, and there is a sensation of vertigo immediately before the loss of consciousness, and at the time of its restoration. The face usually becomes pale first and then red, or either of these conditions may occur without the other being observed.

The spasms may be very slight. Sometimes there is momentary strabismus, at others retraction of the angles of the mouth on one or both sides, rotation of the head or a sudden drawing of it backward, or the tongue is thrust forward and the jaws close on it, inflicting slight injury. Again, the chair in which the patient may be sitting is pushed back with some force, and the body is bent forward, or the muscles of the neck may be affected, and the circulation thus interrupted in the veins of the neck, causing a dark hue of the complexion.

Sometimes the spasms have an appearance of being volitional. A patient under my charge tugs violently at his hand; another walks about the room, but without taking any determinate course; a young lady leaves her chair and stands upon another one at some distance from her, and another talks all kinds of gibberish. My experience of such cases is in accordance with that of Reynolds,[1] to the effect that there is no recollection of these acts. These attacks are often preceded by prodromata of various kinds. The duration rarely exceeds a minute, and is generally much less.

3. *Unconsciousness, with General Tonic and Clonic Convulsions.*—Prodromata may or may not be present. In any event the paroxysm occurs suddenly. The first circumstance may be a cry of a very peculiar character, somewhat resembling the bleating of a young lamb. The eyes become fixed, and the patient falls to the ground, usually with a bound, as if he is shot. The loss of consciousness occurs with the cry, or with the fixedness of the gaze.

The muscles are now thrown into a state of tonic contraction; the respiration is impeded, or altogether arrested; the face, if at first pale, becomes dark; the pupils are dilated, and sensibility is entirely abolished.

Careful examination of a patient in this stage of the paroxysm reveals some important features: the body is rigid, but is usually inclined more to one side than the other, in the position of a tetanic patient with pleurosthotonos; the eyes are open, and are twisted to one side; the face is likewise more retracted on one side than the other; the sterno-cleido-mastoid muscles, and others of the neck, stand out like thick cords; the carotids throb with force; the veins of the head and neck are turgid with black blood, and the pulse is usually weak and fluttering.

After this stage has lasted for a period varying from two or three seconds to half a minute, a great change ensues. The unconsciousness continues, but the general tonic spasm relaxes, and clonic convulsions take its place. These are general, but are ordinarily more strongly marked on one side of the body than on the other. The muscles of the face are alternately contracted and relaxed; the tongue is often thrust between the teeth, and, the jaws being closed upon it, it is terribly injured; the upper and lower extremities are in a state of continued agitation, and the contents of the bladder, rectum, and vesiculæ seminales, may be evacuated.

---

[1] "System of Medicine," vol. ii., p. 261, article "Epilepsy."

The respiration is forced and irregular, froth issues from the mouth, and, if the tongue has been bitten, it is colored with blood.

The muscles of the neck do not relax to any considerable extent; consequently the veins remain distended, and the face continues to be livid. The pupils oscillate, sometimes being dilated and then contracted, or one may be contracted and the other dilated. The heart beats with great irregularity, both as to force and frequency.

This stage may last from a few seconds to five minutes. Cases of longer duration are on record, but they are exceedingly rare.

The third stage of the paroxysm is characterized by the gradual return of consciousness. The patient, though still somewhat convulsed, looks around him, and gives evidence of returning sensibility in other ways. The pupils cease their disorderly movements, and are contracted; the respiration and pulse become more regular, and he may even attempt to speak. It often happens that little spots of extravasated blood make their appearance under the skin of the forehead, eyelids, cheeks, and sometimes on the neck and breast. These disappear in a few days.

The duration of this stage is from a few seconds to four or five minutes, and it is often so slightly marked as to escape observation.

With the cessation of the convulsive movements the stage of stupor usually supervenes, though it may be entirely absent, especially in old cases of epilepsy. During this stage there are sometimes clonic spasms of no great degree of severity. It may last a few minutes or several hours. When the patient arouses from it, he generally has headache, and a feeling of lassitude and soreness of the muscles, from the violent contractions they have undergone.

4. *Irregular or Aborted Paroxysms.*—In these it may happen that the loss of consciousness is not complete, or that the patient has convulsive movements partial in character and accompanied simply by vertigo, or he may have unconsciousness lasting for an hour or more, during which he performs automatic acts, of which he has no recollection, but which are not accompanied by any movements that can properly be called spasmodic.

In his interesting lecture on "Apoplectiform Cerebral Congestion," Trousseau[1] cites a number of cases which were clearly instances of irregular or abortive epileptic paroxysms. Among them is that of a magistrate whose sister was an inmate of a lunatic asylum. He was president of a provincial tribunal. One day he got up all of a sudden, muttered a few unintelligible words, and went to the deliberating-room. The usher followed him, and saw him make water in a corner. A few minutes afterward he returned to his seat, and again listened with intelligence and attention to the pleadings momentarily interrupted. He had no recollection of the incredibly incongruous act he had committed. This gentleman belonged to a literary society, which held its meetings at the Hôtel-de-Ville, of Paris. At one of these, during the discussion of an important historical point, he was seized with vertigo. He ran quickly down to the Place de Hôtel-de-Ville, and walked about for a few minutes on the quays, avoiding with success both carriages and the passers-by. On recovering himself he perceived that he had come out without his great-coat and his hat. He therefore returned to the meeting, and resumed with a perfectly lucid mind the historical discussion in which he had already taken a very active part. He retained no recollection whatever of what had occurred between the beginning of the attack and the moment he recovered himself.

---

[1] *Op. cit.*, Bazire's translation, pp. 19, *et seq.*

Many cases similar to these might be cited from other authors. From a number which have happened in my own experience I adduce the following:

J. H. consulted me for epilepsy in the summer of 1869. His ordinary attacks were of the fully-developed form; but upon two occasions they were different from any with which he had previously been affected. On one of these, while overlooking some workmen, he was observed to put his hand to his head, and then sudddenly to run toward a fence, which he speedily climbed. Jumping down into the back-yard of the adjoining house, he seized a stick of wood near by, and made a furious onslaught on the door and windows. While thus engaged he was seized by several men, and forcibly held, notwithstanding his struggles. While thus being restrained he recovered his consciousness, but had no recollection of any thing which had taken place after he had put his hand to his head, which action he said was due to severe pain with vertigo. The duration of the attack was not over three minutes.

On the other occasion he was seized with pain and vertigo while engaged in paying a bill at a coal-yard. He rushed into the street, and began to turn rapidly round. He was seized and held till he recovered his consciousness. This attack lasted about four minutes.

Subsequently he had a similar paroxysm in my consulting-room. His face suddenly became very pale, his eyes were fixed, and his pupils oscillated. Suddenly he rose from the chair, grasped the mantel-piece for an instant, and then rushed violently around the room, throwing his arms about, and uttering a peculiar inarticulate cry. I made no attempt to restrain him, and in about two minutes he became calm.

During the whole paroxysm his face was pale, and at its close the pupils were dilated. He had no recollection of any thing which had occurred after he rose from the chair, but was conscious then of vertigo.

Another case is that of a girl brought to my clinic at the Bellevue Hospital Medical College during the summer of 1869. She had been severely injured in the skull by a fall against a mass of old iron. Necrosis subsequently ensued, and several large pieces of the external table were exfoliated. While before the class, she started to her feet, and walked several times around the closed area. She was unconscious, and to all appearance insensible. When the paroxysm was over she returned to her seat. The duration did not exceed a minute, and there was no excitement or delirium.

Another patient, a partner in an extensive mercantile establishment, who was subject to attacks of both the *grand* and *petit mal*, left his office at about eleven o'clock for the purpose of getting a signature to a paper of some kind from a gentleman whose place of business was a few minutes' walk distant. Not returning by three o'clock, inquiry was made, and it was ascertained that he had visited the office, obtained the signature, and had left in apparently good health before half-past eleven. Since then nothing had been heard of him. He did not make his appearance at his own office till nearly five o'clock.

The last thing he recollected was passing St. Paul's Church at the corner of Broadway and Vesey Street, just as the congregation was coming out after morning service. It was subsequently ascertained that he had gone to Brooklyn after getting the signature he wanted, had visited a newspaper-office and purchased a paper; had returned to New York, entered an omnibus at the Fulton Ferry, left it at the corner of Twenty-third Street and Fifth Avenue, entered the Fifth Avenue Hotel, and while there recovered his recollection.

But none of these cases, nor any of which I have seen any report, are equal in interest to one which occurred in my practice during the autumn of 1875. The patient, who was engaged in active business as a manufacturer, left his office at about 9 A. M., saying he was going to a florist's to purchase some bulbs. He remained absent eight days. He was tracked all over the city, but the detectives and friends were always an hour or more behind him. It was ascertained that he had been to theatres, to hotels, where he slept, to shops where he had made purchases, and that he had made a journey of a hundred miles from New York, and, losing his ticket and not being able to give a satisfactory account of himself, was put off of the train at a way-station. He had then returned to New York, passed the night at an hotel, and on the eighth day, at about ten o'clock, made his appearance at his office. He had no recollection of any one event which had taken place after leaving his place of business, eight days previously, till he awoke on the morning after his return to the city, and found himself in an hotel at which he was a stranger. It was ascertained beyond question that in all this time his actions had been entirely correct to all appearance, that his speech was coherent, and that he had acted entirely in all respects as any man in the full possession of his mental faculties would have acted. He had drunk nothing but a glass of ale, which he took with some oysters at a restaurant in Sixth Avenue.

It could not be ascertained that this patient had ever had an epileptic paroxysm; but he had a year previously been under my charge for cerebral symptoms, indicating the existence of chronic basilar meningitis, and only a week before his disappearance I had discharged him cured, after a month's treatment for severe pain in the head, dizziness, paralysis of the third nerve on the right side, and extreme insomnia. There were all the indications of specific cause, and I had treated him with large doses of the iodide of potassium, as on the former occasion.

Most, if not all, of the cases of "double consciousness" that have been reported are doubtless epileptic in character. An interesting case of the kind has been related by M. Azam.[1] It is that of a young woman who, after having suffered from hysteria and convulsions, had two distinct phases of existence, living, in fact, two separate and different lives, and exhibiting different likes and dislikes and mental characteristics.

Another case was that of a sergeant, reported by Dr. Mesnet,[2] who, after receiving a severe wound of the skull, had paroxysms characterized by total change in his mentality, and obliviousness of all acts performed in his normal state. During these periods he was conscious, and acted in a logical and coherent manner.

5. *Unconsciousness with Hallucinations.*—In this form of epilepsy, which I described in a paper read before the American Neurological Association, June 18, 1880,[3] the characteristics are conscious hallucinations, followed by unconsciousness but unattended by muscular spasm. I have had the opportunity of seeing two cases (one since the reading of the paper) while the paroxysms were present, and in neither was there the least spasmodic action. I quote part of the description of one of the cases, that of a young woman on which the memoir in question is based:

"I had the opportunity of witnessing seventeen paroxysms. Sometimes they were preceded by a well-marked aura, and this was always a sensation apparently somewhere within the cranium, but not capable of being exactly localized or described. This was never felt until within the last two years. It lasted only a second or two, and was immediately followed by the 'vision.'

---

[1] "Amnesie périodique, ou dédoublement de la vie," *Annales Médico-psychologiques*, July, 1876.

[2] *Union Médicale*, July 21 and 23, 1874. Translated in *The Chicago Journal for Nervous and Mental Disease*, January, 1875.

[3] "On Thalamic Epilepsy," *Archives of Scientific Medicine*, August, 1880. Also, *Neurological Contributions*, No. III, 1881.

"The first paroxysm of this series which I witnessed was ushered in by the aura. She had hardly time to say, 'It's coming,' when the hallucination began. She described it as consisting of a large white bear in motion before her on the carpet. It seemed to be walking slowly to and fro, its head bent toward the floor as if scenting something. I closely watched her, and could detect no spasm anywhere. She spoke clearly, without hesitation, and with entire distinctness. The pupils were normal.

"I had taken out my watch to time the duration of the attack. Thirty-five seconds elapsed, and then her pupils suddenly dilated, her head fell forward, and her left hand, which was at this instant pointing in the direction of the visional bear, dropped to her side. I pinched the skin of her face, then of each hand, without eliciting any evidence of cutaneous sensibility. I took up a fold of skin on each forearm just above the wrist and stuck a cataract-needle, which was at hand, through it, with a like result. Her pulse—I had not felt it during the existence of the hallucination—was beating at the rate of about sixty a minute, and was full. Her face had not altered in color, nor was there any other change in it except such as was due to relaxation of the muscles—such as is present in sleep. The eyelids were closed, but not spasmodically. She remained in this state exactly twenty-eight seconds, breathing perhaps a little more slowly and deeply than before the accession of the paroxysm. Suddenly she raised her head, looked inquiringly around her for a moment, and then, as if becoming aware of a sensation, looked at both her arms where I had pricked them. A drop of blood was oozing from each puncture. She asked what it was, and then, without waiting for an answer, exclaimed, 'You have bled me!' She was then entirely herself, and talked coherently, and without the least excitement, about the hallucination.

"While making memoranda of the phenomena I had observed, and while she was walking up and down the floor, she said that she was going to have another attack, as she felt the peculiar sensation again in her head. She had no sooner uttered the words than the vision came. 'It's a girl this time!' she exclaimed—'a girl with long auburn hair, and a cap on her head; she looks like a French nurse. I think I will sit down, for if I do not I shall fall as soon as I become insensible;' saying which, she quietly sat down in a large arm-chair.

"I pinched the skin of her right hand. 'Oh!' she exclaimed, 'I feel that; I am not insensible yet; I see everything in the room as well as I do the girl who is not here. I can feel the least touch, and my hearing is as good as ever.'

"I asked her what 'the girl' was doing. 'Oh, nothing,' she replied: 'she is only standing there in front of the fireplace, looking at me.'

"I told her to shut her eyes, and then to tell me if she still saw 'the girl.'

"'Yes,' she answered, 'just as distinctly as I did when they were open.'

"At forty-one seconds she became unconscious, and remained in this state for one minute and five seconds, awaking—I say awaking, for her appearance was like that of a person asleep—suddenly, and apparently in a normal condition of mental and physical health."

Subsequently, from inattention to treatment on the part of the patient, the paroxysms passed into others with strong muscular contractions, and she exhibited indications of a tendency to the perpetration of acts of violence.

In all, six cases of this interesting form of epilepsy have come under my observation; and I have learned of others being recognized by several physicians of competent powers of observation.

Relative to the mental disturbance which sometimes ensues upon epileptic paroxysms, Dr. Hughlings Jackson[1] has recently given some interesting details relative to acts performed by epileptics during periods of unconsciousness. In his opinion such acts are automatic, not—to speak exactly—epileptic, but post-epileptic. "The condition after the paroxysm is duplex: (1) there is loss or defect of consciousness, and there is (2) mental automatism. In other words, there is (1) loss of control, *permitting* (2) increased automatic action." The epileptic seizure may be so slight and transitory as to escape observation, but the slighter it is the more apt is the resulting automatism to be complex and elaborate.

Dr. Jackson gives a number of exceedingly interesting cases in illustration of his views, which in addition are enforced with much cogent reasoning. But, while in the main agreeing with him, I am scarcely prepared to deny that such unconscious attacks may not be substituted for the more fully-developed paroxysm instead of, as in his opinion, always following a seizure.

Epileptic fits may take place at night during sleep, and the patient be unaware of their existence, unless he inflicts some injury on himself, such as biting his tongue, or is told of their occurrence by persons who may be in the same room with him. In two hundred and six of my cases the period of access is noted, and, of these, forty-seven were nocturnal, and one hundred and fifty-nine diurnal.

In the intervals between the paroxysms epileptics often exhibit certain evidences of disordered mental, sensorial, and motor functions. Thus, as regards the first category, the memory may be impaired, and there may be diminished mental power. There are, however, many exceptions to this rule; and, even where there have been a great many attacks, the mind may preserve its normal degree of integrity. As

---

[1] "On Temporary Mental Disorders after Epileptic Paroxysms," "West Riding Lunatic Asylum Medical Reports," vol. v., p. 105.

Reynolds remarks, in regard to this point: "A patient may be epileptic and a lunatic; he may be epileptic and asthmatic, but there are some epileptics whose minds are as healthy as their lungs; and, so far as the natural history of epilepsy is concerned, it is a mistake to derive it from complicated cases." Still, in the majority of cases, it will be found that the mind sooner or later becomes involved, and it sometimes happens that a single attack causes marked intellectual deterioration.

Derangements of sensibility are common from the beginning. Headache, a feeling of constriction around the forehead, and occasionally a pain at the back of the head, are noticed. Vertigo is also frequently present, as are also sensations of numbness in different parts of the body. The pupils are almost invariably dilated.

The motor power of the patient is generally weakened without there being any decided paralysis. Twitchings of the muscles are not uncommon, and there is often a general excitability of the reflex faculty of the spinal cord, by which jerkings of the limbs are produced by slight excitations.

The circulation is generally sluggish, the extremities are cold, and the capillaries are turgid and inactive, so that, if the finger be pressed firmly upon the skin, a considerable period elapses before the white spot disappears by the refilling of the vessels.

In examining with the ophthalmoscope the fundus of the eye in epileptics, we can often detect evidences either of cerebral congestion or of anæmia, and thus obtain valuable indications for treatment. For several years, in my lectures, I have constantly insisted on this point, and in my cliniques have exhibited several cases in which I had been guided to successful treatment by the ophthalmoscope. Drs. Köstle and Niemetshek,[1] of Prague, consider that the brain in epileptics is always anæmic, and that this condition is invariably found by ophthalmoscopic examination. According to these observers, the venous pulse is produced when the eye is made anæmic, and they assert that the retina is anæmic, and that there is consequently venous pulsation in every case of epilepsy. That this opinion is erroneous, both as to the facts and inferences, I am very sure. Venous pulsation, so far from being indicative of anæmia, really shows the existence of the very opposite condition. My observations are, however, to the effect that venous pulsation is present in many cases of epilepsy, and that it accompanies dilatation of the veins.

There is no invariable rule relative to the occurrence of any particular form of epilepsy in the same person. It thus often happens that all the varieties of paroxysm mentioned, except the irregular or aborted form, which is more rare, are met with in one individual. The more severe forms may occur at longer intervals, and the milder forms more frequently. As regards frequency, there are great variations. Some patients go a year or more without attacks, while others have several every day. It generally happens that the intervals become progressively shorter. As a rule, attacks of the milder forms are more frequent than the fully-developed paroxysm, and attacks of the latter are milder, as they are more frequent.

---

[1] *Prager Vierteljahrschrift*, H. 106, 107, 1870, and *Quarterly Journal of Psychological Medicine*, January, 1871, p. 128.

Mania is sometimes a consequence of epilepsy. It comes on after the attack, and is rarely of more than a few minutes' duration. Those cases in which it precedes the paroxysm, and lasts several hours or days, are cases of mania conjoined with epilepsy—a combination which, as every insane asylum shows, is not uncommon. The mania of epilepsy is usually of a very exalted character, and during its existence the subject may commit homicide or other crimes.

The mental state of epilepsy has been well studied by Falret,[1] and a very interesting case has been recently reported by Dr. Thorne,[2] in a paper entitled "Masked Epilepsy." In this instance the patient often returned to his home without being able to give any account of what he had been doing or where he had been. During these attacks he was frequently the subject of that form of mental derangement called kleptomania. Generally they ensued on paroxysms either of the *grand* or *petit mal*, but sometimes they were substituted for the regular seizures. He had no recollection of what occurred during the attacks. Sometimes he was furiously excited in them, and would endeavor to injure himself and others in his blind rage.

Relative to the diagnosis of the remarkable paroxysms, the main feature of which is unconsciousness, or rather non-recollection of consciousness, in which the individual acts apparently automatically, great difficulties exist. Probably nothing short of a full history of the case, from infancy up, will suffice for the recognition of the real nature of the phenomena. There appears to be an idea in the minds of some physicians, that every outrageous criminal act is the result of epilepsy, and so wide-spread is this notion, that now the first plea of the murderer is, that he "knew nothing about it;" and the fact that an individual who has perpetrated a murderous outrage is the subject from time to time of epileptic seizures, is regarded as sufficient to absolve him from all responsibility for his actions. The fact of a discolored spot on his pillow, or of an infantile convulsion, is seized upon as a valid reason for acquittal, or even for setting aside a verdict found after a full and fair trial. In the first place, it must be understood that an undoubted epileptic is just as capable of murdering for revenge or gain as is a healthy person, and that he is just as accountable, and should accordingly suffer the full penalty of the law for his conduct. At the same time, it is not to be questioned that acts of violence may be perpetrated during seizures which are either epileptic or the direct consequence of an epileptic paroxysm. It is only by the most thorough and careful inquiry into all the motives for and circumstances attending upon the act, as well as all the antecedents of the individual, that a proper discrimination can be made. Each case must be determined for itself; there are no rules applicable invariably to all.

---

[1] "De l'état mental des épileptiques," *Archives générales de médecine*, Décembre, 1860, et Avril et Octobre, 1861.

[2] "St. Bartholomew's Hospital Reports," 1870.

The medico-legal relations of epilepsy do not, however, come within the scope of the present treatise.

Paralysis may follow epilepsy, but, unless the case is complicated with some organic disease of the brain or spinal cord, the loss of power is temporary.

Causes.—Among the predisposing causes of epilepsy, hereditary tendency stands first. Reynolds [1] states that, in about one-third of the cases under his observation, hereditary taint existed. He does not, by this statement, however, mean to assert that epilepsy existed in one-third of the parents, but that some disease of the nervous system, more or less closely allied to epilepsy, was present in either the parents, the grandparents, the aunts, uncles, brothers, or sisters. Only twelve per cent. of his cases gave a distinct history of epilepsy in either branch of their families.

Herpin,[2] of sixty-eight cases, found that ten were descended from epileptic ancestors.

Delasiauve,[3] of three hundred cases, found decided evidence of hereditary tendency in thirty-three. In one hundred and sixty-seven there were no data, and in one hundred and twenty hereditary taint was denied. Of the thirty-three cases, five were descended from epileptic ancestors.

Sieveking [4] found that hereditary influence was present in 11.1 per cent. of his cases.

In my own experience I have notes in regard to this point in three hundred and ninety-six cases. Of these, sixty-four had epileptic fathers, mothers, grandparents, uncles, aunts, brothers, or sisters, and forty-eight had relatives insane, hysterical, cataleptic, affected with severe neuralgia, or of remarkably irritable nervous systems.

Sex does not appear to exercise any appreciable influence as a predisposing cause. Of five hundred and seventy-two cases noted by myself, two hundred and ninety-eight were in males and two hundred and seventy-four in females. Other authors have, however, had directly opposite experience.

Age has a very decided influence. Reynolds gives the following table of one hundred and seventy-two cases collected by himself:

---

[1] *Op. cit.*, p. 253.

[2] "Du pronostic et du traitement curatif de l'épilepsie," Paris, 1852, p. 325.

[3] *Op. cit.*, p. 189.

[4] "On Epilepsy," etc., London, 1858, p. 74.

Age at Commencement.	Males.	Females.	Total.
Under 10 years	10	9	19
Between 10 and 20 years	66	40	106
Between 20 and 44 years	25	20	45
Over 45 years	1	1	2
Total	102	70	172

My own cases were as follows :

Age at Commencement.	Males.	Females.	Total.
Under 10 years	31	29	60
Between 10 and 20 years	178	151	329
Between 20 and 45 years	72	71	143
Over 45 years	17	23	40
Total	298	274	572

It is thus seen that the period of life between ten and twenty years is that at which epilepsy is most apt to occur. The experience of others is to the same effect. The influence of temperament has been thought important by some writers. But, aside from the different opinions entertained relative to the characteristics of the temperaments, it is by no means established that, even when strictly defined, temperament exercises any effect as a predisposing cause. I have no accurate records on this point, though so far as my memory serves me I have observed no marked predominance of epileptics with any temperament.

The exciting causes may very properly be classified as psychical, eccentric, general organic changes, and physical influences. Relative to the influences of these causes, Reynolds gives the following table :

Nature of Cause.	No. of Cases.
I. Psychical—such as fright, grief, worry, overwork	29
II. Eccentric irritation—dentition, indigestion, venereal excesses, dysentery, etc	16
III. General organic changes—fatigue, pregnancy, miscarriages, rheumatic fever, scarlet fever, diphtheria, pneumonia	9
IV. Physical influences—blows on head, falls, insolation, cuts	9

In my own cases no exciting cause could be assigned in one hundred and seventy-seven. The remaining three hundred and ninety-five cases were, according to the evidence received, caused as follows :

Fright	35
Anxiety	17
Grief	30
Over mental exertion	48
Dentition	21
Indigestion	33
Carried forward	184

Brought forward	184
Venereal and sexual excesses	60
Menstrual derangement	56
Blows on the head	24
Peripheral wounds and injuries	4
Falls	13
Sunstroke	17
Scarlet fever	8
Measles	3
Diphtheria	9
Pregnancy	3
Syphilis	13
Malaria	6
	395

Diagnosis.—The diagnosis of epilepsy presents no difficulties to the careful observer. It may, however, be confounded with several conditions, the principal of which are cerebral congestion, cerebral hæmorrhage, hysteria, the convulsions of infancy and of Bright's disease, poisoning by opium and alcohol, syncope, and with the convulsions of epileptiform character which occur in the course of certain organic diseases of the brain.

The diagnosis from cerebral congestion and cerebral hæmorrhage has already been given in the chapters treating of those affections. In hysteria, the convulsions, which are sometimes epileptiform in character, are preceded or accompanied by other evidences of the hysterical state. Consciousness is rarely entirely lost, the tongue is not bitten, and there is no subsequent stage of stupor.

The convulsions of infancy not epileptic are not repeated but from a readily-ascertained exciting cause, such as dentition, indigestion, falls, etc. So far as the paroxysm is concerned, I know of no specific points of difference; but it must be recollected that the paroxysm is not the only feature of epilepsy, and that it is the only feature of infantile convulsions. These latter may pass into epilepsy; but, if they do not, I have never been able to find a single case in my experience in which epilepsy ensuing in adult life has been preceded by the ordinary infantile convulsions. In Bright's disease, though the convulsions may be epileptiform in character, coma is the principal feature, and the history of the case will further serve to render the diagnosis exact. The same remarks are applicable to poisoning by opium and alcohol.

From syncope epilepsy is distinguished by the facts that the loss of consciousness is sudden and complete, that the pulse is not feeble, and that recovery is rapid. These remarks apply to the milder attacks without convulsions. From the more severe forms of the paroxysm the distinction is too obvious to require amplification.

In organic diseases of the brain, such as tumors, softening, sclerosis, etc., the accompanying symptoms, pain, paralysis, tremor, imbecility, difficulties of speech, and derangements of the special senses, will serve to distinguish them from epilepsy.

Epilepsy is often assumed by designing persons for purposes of fraud. In such cases the pretender usually overacts his part; his sensibility is not abolished, as may readily be ascertained by putting the end of the finger on the conjunctiva, and the size of the pupils is not altered.

Prognosis.—The prognosis depends to a great extent on the duration of the disease. Recent cases can often be cured, but those which have lasted for several years are rarely brought to a favorable termination. Among the other unfavorable elements are the existence of hereditary influence, the beginning of the disease late in life, the presence of material mental weakness, and the existence of long intervals between the attacks.

As regards the probability of the supervention of any form of intellectual derangement or debility, the most important ascertained point is that the mild paroxysms unattended by convulsions are more productive of mental decay than the severe form of seizure. The occurrence of the first attack late in life is likewise a predisponent to dementia.

I have never, in my own experience, known death to take place during a paroxysm of true epilepsy; such cases, however, do occur. Usually, some intercurrent affection carries the patient off, though even with this liability life is sometimes astonishingly prolonged. I am acquainted with the case of a lady who is now sixty-five years of age, and who, since her tenth year, has averaged six paroxysms daily, all of the severest character. Her mind is almost entirely gone, but physically her health is excellent, and to all appearance she may live twenty years longer.

I am not aware of any exact observations tending to show the relative danger to life of attacks of the milder and severer forms; though it is reasonable to suppose that, so far as regards the occurrence of death during the paroxysm, the convulsive form is more fatal.

Morbid Anatomy.—In post-mortem examinations of persons dying epileptic, abnormal conditions are found in every part of the brain and spinal cord. Some of these lesions are undoubtedly secondary, others unessential, while those which may be considered primary vary in their seat and character. In a great many cases, perhaps the majority, no lesions are discoverable.

No one has been more thorough in the search for the essential cause of epilepsy than Schroeder van der Kolk;[1] though his observations can scarcely be regarded as yielding conclusive results, they serve to show, when taken in connection with the pathology of the disease in question, that its seat is mainly in the medulla oblongata, with secondary implication of other parts of the cerebro-spinal nervous system. Oftentimes, in accordance with other pathologists, he found nothing to account for the affection, but at others he found hardening and contraction of the medulla oblongata, and again degeneration of the brain either as a consequence or cause of the disease. Microscopical examination sometimes showed him the medulla indurated, sometimes softened, and, as a constant phenomenon, "whether the patient died in or out of the fit, great redness and vascular tension in the fourth ventricle, penetrating into the medulla oblongata sometimes to a considerable depth." These appearances were due to enlargement of the blood-vessels, as was shown by microscopical measurements. It is probable, however, as Schroeder van der Kolk asserts, that the lesions in question are the results, and not the causes, of the paroxysms. Still they suffice to indicate the main seat of the disease to be the medulla oblongata.

---

[1] "On the Minute Structure and Functions of the Medulla Oblongata, and on the Proximate Causes and Rational Treatment of Epilepsy," "New Sydenham Society Translations," London, 1859.

Other observers have not so uniformly found this enlargement of the blood-vessels of the medulla. In three cases of death occurring in epileptics, in which I have had the opportunity of making post-mortem examinations, they certainly did not exist, nor was there any other lesion detected by the most careful microscopical exploration. In one other case the vessels of the medulla oblongata were enlarged, and there was amyloid degeneration of the pituitary body.

Fox[1] gives the following list of the post-mortem appearances:

Foreign bodies developed on the meninges, in the ventricles, in the cortical substance; increase of subarachnoid fluid or distention of the ventricles by serum, induration, softening, and general swelling of the cerebral mass; general or partial hyperæmia, cysts, tubercles, cancers, exostoses, periosteal growths, thickening, or some change of the arachnoid or the pia mater; abnormal thickness or abnormal thinness of the cranial bones; excessive size of head, increase of the volume of the cranial cavity, deformities or abnormality in the conformation of this cavity; caries of the cranial bones; pus between the bone and the dura mater; acute or chronic hydrocephalus, hydatids, ossification of the dura mater, tubercle of the dura mater or pachymeningitis, abscess in the cerebral tissue, spots or regions of hæmorrhage; various traumatic lesions; alterations of the pineal gland; inequality of weight and size of the cerebral hemisphere; various lesions connected with blood-vessels—aneurism, embolism, atheroma, increase in size of the capillaries in the medulla oblongata, fatty degeneration of some portion of the medulla oblongata; capillary dilatation in the pons and cerebellum; hæmorrhage of pons; anæmia of brain, either from disease of vessels or dependent upon general anæmia, etc., etc.

---

[1] "The Pathological Anatomy of the Nervous Centres," London, 1874, p. 305.

Indeed, no point is more thoroughly established than that epilepsy results from very different morbid conditions, and that they are simply the starting-points in the majority of cases. The true lesion has not yet been detected, and in fact, as we shall presently see when discussing the pathology of the disease, there may be no necessary anatomical lesion whatever.

Pathology.—The points which may be considered as to some extent established relative to the pathology of epilepsy are briefly summarized as follows by Reynolds :[1]

"1. That the seat of primary derangement is the medulla oblongata and upper portion of the spinal cord.

"2. That the derangement consists in an increased and perverted readiness of action in these organs, the result of such action being the induction of spasm in the contractile fibres of the vessels supplying the brain, and in those of the muscles of the face, pharynx, larynx, respiratory apparatus, and limbs generally.

"By contraction of the vessels the brain is deprived of blood, and consciousness is arrested; the face is or may be deprived of blood, and there is pallor; by contraction of the vessels which have been mentioned, there is arrest of respiration, the chest-walls are fixed, and the other phenomena of the first stage of the attack are brought about.

"3. That the arrest of breathing leads to the special convulsions of asphyxia, and that the amount of these is in direct proportion to the perfection and continuance of the asphyxia.

"4. That the subsequent phenomena are those of poisoned blood, i. e., of blood poisoned by the retention of carbonic acid, and altered by the absence of a due amount of oxygen.

"5. That the primary nutrition-change, which is the starting-point of epilepsy, may exist alone, and epilepsy be an idiopathic disease, i. e., a *morbus per se*.

"6. That this change may be transmitted hereditarily.

"7. That it may be induced by conditions acting upon the nervous centres directly, such as mechanical injuries, overwork, insolation, emotional disturbances, excessive venery, etc.

"8. That the nutrition-changes of epilepsy may be a part of some general metamorphosis, such as that present in the several cachexiæ—rheumatism, gout, syphilis, scrofula, and the like.

"9. That it may be induced by some unknown circumstances determining a relative excess of change in the medulla during the general excess and perversion of organic change occurring at the periods of puberty, of pregnancy, and of dentition.

---

[1] *Op. cit.*, p. 275, and more fully stated in his "Treatise on Epilepsy, its Symptoms, Treatment, and Relations to other Chronic Convulsive Diseases," London, 1861, chapter v., p. 238.

"10. That it may be due to diseased action, extending from contiguous portions of the nervous centres or their appendages.

"11. That the so-called epileptic aura is a condition of sensation or of motion, dependent upon some change in the central nervous system, and is, like the paroxysm, a peripheral expression of the disease, and not its cause."

While admitting the correctness of these conclusions, they do not, in my opinion, tell the whole story of the theory of epilepsy. In very many memoirs Dr. Brown-Séquard has pointed out the dependence of the affection upon injuries of the upper part of the spinal cord, and upon irritations existing in various parts of the body. His researches, and facts observed every day by physicians who see many cases of epilepsy, show very conclusively that the starting-point is often in the sympathetic nerve—the nerve by which the calibre of the blood-vessels is regulated.

Neither can I accept the view that the first intra-cranial condition producing a paroxysm is in all cases spasm of the blood-vessels and the consequent deprivation of the blood-supply to the brain. On the contrary, I am very sure that the primary state is often paralysis of the cerebral blood-vessels and resulting hyperæmia. By this condition the medulla oblongata is thrown into a state of over-excitation, giving rise to convulsions, and consciousness is lost from the fact that the hemispheres participate. That convulsions, epileptiform in character, may be produced both by cerebral anæmia and cerebral hyperæmia, when either condition involves the medulla oblongata, is a fact which experiment has abundantly established, and that loss of consciousness follows either condition involving the hemispheres is equally certain. We have, consequently, two kinds of epilepsy—the one due to anæmia, the other to congestion—and it is to this fact that is due the circumstance that sometimes the paroxysms are prevented by measures which tend to increase the amount of blood in the brain, and at others by remedies which exercise a contrary influence. The existence of the two species of epilepsy is likewise shown by ophthalmoscopic examination—a point upon which I have already insisted.

During natural sleep the amount of blood is, as I have elsewhere shown, decreased from the quantity which circulates in the cerebral blood-vessels during wakefulness. Epilepsy occurring during sleep is therefore of the anæmic variety. But it often happens that sleep passes gradually into stupor, from the fact that causes tending to increase the flow of blood to the brain, or to arrest its passage from this organ, are in operation. In such cases epilepsy of the congestive variety may be induced.

In those cases in which the tongue is bitten, the medulla oblongata is probably always in a condition of hyperæmia; and this state, as Schroeder van der Kolk has very conclusively shown, is mainly in the course of the roots of the hypoglossal nerve. The intermissions between the attacks are ingeniously explained by the same able observer, by likening the cells of the medulla oblongata to Leyden-jars charged with electricity, or to the electrical organs of the conger-eel and torpedo. After being discharged, time is necessary for the reaccumulation of sufficient electricity to discharge them again; and, when the cells of the medulla have once discharged themselves in an epileptic convulsion, a period must elapse before another access can take place.

Nothnagel[1] believes that the pons Varolii and the medulla oblongata are the seat of epilepsy, and that it is in these centres that we are to look for the anatomical changes. Although, as his own experiments as well as those of Hitzig show that epilepsy may be produced by irritation of the cortical substance of the brain, the fact only proves that such irritation is an exciting cause, and is no more to be regarded as indicating the cortex as the seat of the disease than the fact that irritation of the sciatic nerve, followed by epilepsy, indicates that part of the nervous system as containing the essential lesion.

The foregoing remarks apply in the main to that form of epileptic seizure characterized by convulsion. In the imperfectly-developed attacks the implication of the medulla oblongata must be very slight, the hemispheres being the organs mainly affected, and the condition being sometimes anæmic, at others hyperæmic.

It must not be supposed, from what has been said, that simple cerebral anæmia and simple cerebral congestion, attended with epileptiform convulsions, are identical with the anæmia and congestion of epilepsy. This disease is cerebral anæmia or congestion with another element, the exact nature of which we do not understand, but which is certainly of such a character as to constitute the main differential point between epilepsy and any other affection.

A chapter on epilepsy would be manifestly incomplete without a statement of the views held by Dr. Hughlings Jackson[2] relative to its pathology and natural history. According to this eminent authority those parts of the body suffer first and most, from convulsions or other manifestations of the disease, which are most frequently brought into volitional action, and those parts least which are most automatic in their operation.

Thus he says, in a paper published in the *Lancet*, February 1, 1873:

"There are three parts where fits of this group mostly begin: (1) in the hand; (2) in the face, or tongue, or both; (3) in the foot. In other words, they usually begin in those parts of one side of the body which have the most voluntary use. The order of frequency in which parts suffer illustrates the same law. I mean that fits beginning in the hand are commonest; next in frequency are those which begin in the face or tongue, and rarest are those which begin in the foot. The law is seen in details. When the fit begins in the hand, the index-finger and thumb are usually the digits first seized; when in the face, the side of the cheek is first in spasm; when in the foot, almost invariably the great-toe."

---

[1] "Epilepsie," in Ziemssen's "Handbuch der speciellen Pathologie und Therapie," zwölfter Band, "Krankheiten des Nervensystems," ii., zweiter Hälfte, pp. 250, *et seq.*

[2] "On the Anatomical, Physiological and Pathological Investigation of Epileptics," "West Riding Lunatic Asylum Medical Reports," vol. iii., 1873, p. 315.

As Dr. Jackson says, the spasm "prefers," so to speak, to begin in those parts which have the most voluntary uses; in other words, in those parts which have the more leading, independent, separate and varied movements; in other words still, in those parts the movements of which are last acquired—"educated." Physiologically, a voluntary part, the hand, for instance, is one which has the greater number of *different* movements at the greater number of different intervals; that is, the more "varied" uses. An automatic part, the chest, for example, is one which has the greater number of similar movements at the greater number of equal intervals; shortly, the more "similar" uses. Hence, convulsions which begin in the hand usually begin in the thumb and index-finger—in the most voluntary parts of the body.

An epileptic paroxysm is a sudden, excessive, and rapid discharge of gray matter of some part of the brain. Instead of working off its force gradually and regularly, it explodes it, as it were. The gray matter which is the seat of a "discharging lesion" is in a permanently abnormal state of nutrition, and hence is permanently abnormal in function. Thus a first fit is a discharge of a part which has for some time before been in a state of mal-nutrition. And a still further inference is that such "causes" of epilepsy as fright are only determining causes of the first explosion.

In regard to this latter point, I am entirely in accord with Dr. Jackson. We frequently see cases of epilepsy which, we are told, were originally caused by a mental shock of some kind. But if the shock were in reality the primary cause there should be no subsequent epileptic seizures. With the cessation of the cause the effect should cease. On the contrary, we find that after some time, generally quite long, which of itself is sufficient to show that the continuance is not due to the initial convulsion, a second occurs, and then, after a shorter interval, a third, and so on. It is very evident that if the fright were the cause the fits would be more frequent at first, and less so subsequently.

But to return to Dr. Jackson's views: "Epilepsy is not a particular grouping of symptoms occurring occasionally; it is a name for any sort of nervous symptom or group of symptoms occurring occasionally from local discharge, whether the discharge puts muscles in movement or not—that is, whether there be a convulsion or not matters nothing for the definition. A paroxysm of subjective sensation of smell is an epilepsy as much as is a paroxysm of convulsion; each is the result of sudden local discharge of gray matter.

"It does not matter for the definition whether there be loss of consciousness or not; loss of consciousness is a fundamental thing in most of the accepted definitions. If there be no loss of consciousness there is, according to most physicians, not epilepsy, and the term 'epileptiform' is used. But, even when using the term epilepsy in the ordinary sense of the word, the separation into cases where there is, and where there is not loss of consciousness, has no physiological warrant. It is an arbitrary distinction of psychological parentage. Loss of consciousness is not an utterly different thing from other symptoms. It is not to be spoken of as an epiphenomenon or as a complication. Consciousness has of course anatomical substrata as much as speaking has. The sensori-motor processes concerned in consciousness are only in degree different from others. They are the most special of all special nervous processes, the series evolved out of all other (lower) series.

"To lose consciousness is to lose *the use of the most special of all nervous processes* whatsoever. If those parts of the brain be first affected by strong discharge where the most special of all nervous processes lie, there will be loss of consciousness *at the outset.* If processes of subordinate series be discharged, loss of consciousness, of course, occurs later. For example, in cases of convulsions beginning in the hand, consciousness is in most cases lost as soon as or just before the leg is reached by the spasm. In these cases the internal process will be that consciousness is lost as soon as the most special of all processes are reached by the internal discharge (or since the sensori-motor processes underlying consciousness are evolved out of lower series), when as large a quantity of a subordinate yet important series is put *hors de combat.* But, of course, one does not locate consciousness so geographically as the mere words we must use seem to imply. If a patient suddenly loses, by any process, the use of any *large part* of either of the two *highest* divisions of the nervous system, he will lose consciousness.

"The following are epilepsies:

"(1) A sudden and temporary stench in the nose, with transient unconsciousness; (2) a sudden and temporary development of blue vision; (3) spasm of the right side of the face with stoppage of speech; (4) tingling of the index-finger and thumb, followed by spasm of the hand and forearm; (5) a convulsion almost immediately universal, with immediate loss of consciousness; (6) certain vertiginous attacks.

"All these six seizures are alike, in that each results from an occasional and excessive discharge of unstable gray matter. This is the one functional alteration of nerve-tissue underlying the different phenomena."

Dr. Jackson then goes on to state that though these six kinds of seizures are alike physiologically, they are very unlike anatomically.

That is, that the seat of the discharging lesion is different for each, and he urges that the efforts of physicians should be directed to the discovery of this seat from a consideration of the character and localization of the manifestation. In a "destroying lesion," such, for instance, as is produced by cerebral hæmorrhage, the scientific physician endeavors, by a careful study of the phenomena, to determine the situation of the injury, but in cases of spasm the inquiries rarely relate to anything more than an attempt to ascertain the character of the convulsion. That this is true is not to be doubted.

Further, Dr. Jackson asserts that by comparing the phenomena produced by a "destroying lesion" with those which result from a "discharging lesion," we obtain very important data for further comparison. Thus, taking the left corpus striatum as the seat of the first or slightest degree of alteration, and then of the second or severer form, and we have :

FIRST DEGREE.

Corpus Striatum Palsy.	*Corpus Striatum Epilepsy.*
Mouth turns to left.	Mouth drawn to right.
Right arm paralyzed.	Right arm convulsed.
Right leg paralyzed.	Right leg convulsed.

SECOND DEGREE.

Corpus Striatum Palsy.	*Corpus Striatum Epilepsy.*
Head turns to left.	Head drawn to right.
Two eyes turn to left.	Two eyes drawn to right.
Face turns to left.	Face drawn to right.
Trunk-muscles weaker on right.	Trunk-muscles in spasm on right (?).
Arm and leg paralyzed on right.	Arm and leg in spasm on right.

But as there is a still higher degree of "destroying lesion," so there is a graver form of "discharging lesion." In the one the corpus striatum of one side may be so injured as to produce paralysis of both sides of the body. In the other a convulsion may originate in the hand of one side, pass over to the opposite side of the body, and become general.

This is a brief statement of Dr. Hughlings Jackson's views on the pathology of epilepsy. Perhaps it would be too much to say that they should be adopted in their entirety, but, that they are in great part correct, every physician who has seen much of the very important disease to which they relate will readily admit. The point in regard to which I should be most disposed to differ with him is that in which he too sweepingly, in my opinion, classes all "occasional, sudden, excessive, rapid and local, sensorial or motor phenomena" as epileptic. Thus, I am quite sure I have repeatedly witnessed "tingling of the index-finger and thumb, followed by spasm of the hand and forearm," result from injury of the eccentric nervous system, from pressure on, or other injury of, the brachial plexus, for instance. Now, although such lesion may, under certain circumstances, produce such intra-cranial disorder as eventually to cause epilepsy, knowing what we do of the functions of the nerves and the effects of injuries to their trunks, we need not go so far as the gray matter for an explanation of the phenomena. Experiments on animals—and indeed as I have repeatedly witnessed in the human subject—show us that, even when a nerve-trunk is divided, irritation of its peripheral extremity will give rise to just such phenomena as Dr. Jackson calls epileptic, except in the one point—not an essential one—of "tingling." In a patient whom I saw in the Presbyterian Hospital a year or so ago, in the service of Dr. Post, the median nerve was exposed for the space of over two inches, and when it was touched with a probe or the finger, tingling in the skin below and spasm of the muscles of the forearm were at once produced.

In the present state of our knowledge it appears to me better to regard no spasm as epileptic, which is not accompanied with loss, or at least disturbance of consciousness. The experiments of Hitzig, Ferrier, and others, certainly throw a great deal of light on the nature of the epileptic phenomena, and give great support to many of Dr. Jackson's arguments; but they also show us that irritation of the gray matter of the brain will cause spasms, which, though partaking to a superficial examination of the character of epilepsy, are clearly not this disease, even as Dr. Jackson regards it. It is true that such irritation repeatedly made will in time so alter the properties of the gray matter as to lead to the production of spontaneous spasms, which may be epileptic, but that is quite a different thing.

The experiments made by Dr. Roberts Bartholow[1] on a patient under his charge, in the Good Samaritan Hospital in Cincinnati, show that both disorders of sensibility and spasm are produced in the human subject by irritation of the gray matter of the cerebral convolutions; but in this case the phenomena disappeared as soon as the irritation ceased. Such transient results, clearly and distinctly due to an irritation of the gray matter, may be epileptiform, but to my mind they are not epileptic.

But quite recently Hitzig[2] has succeeded in producing true epilepsy in animals by irritating the cortical centres; after a shorter or longer period—a day to five or six weeks—spontaneous, well-characterized epileptic convulsions ensued. The importance of such observations as those of Bartholow and Hitzig can scarcely be over-estimated.

Brown-Séquard has shown that epilepsy may be caused by irritation of the peripheral nervous system, and it is quite certain that the tingling and spasm of the hand, which are at first perhaps only due to eccentric lesions or derangements, may result in epilepsy.

A case is now under my charge—a young gentleman from North

---

[1] *American Journal of the Medical Sciences*, April, 1874.

[2] "Untersuchungen ueber das Gehirn," Berlin, 1874, p. 271.

Carolina, whom I saw first over two years since. At that time it was only necessary to touch the left side of his neck, over the middle third of the sterno-mastoid muscle, to induce spasm of the muscles of the neck, shoulder, and face, on the same side, unaccompanied by loss of consciousness. This condition had apparently been induced in the first instance by his wearing a high shirt-collar, and in the beginning consisted of nothing more than a slight twitching of the muscles at the left angle of the mouth. Probably, if he had then ceased wearing that kind of collar, the excessive hyperæsthesia of the eccentric nerves would have spontaneously ceased. As it was, an increase of all the phenomena took place; and finally, the least touch, even that of a camel's-hair pencil or a current of air, was sufficient to induce a spasm. Blistering, cauterization, and all kinds of local anæsthetics, were tried in vain, but eventually they ceased under the use of large doses of the bromide of sodium. But during all this time, unless an irritation of some kind—the lighter the more powerful, for strong pressure was not an efficient agent—there were no spasms. That such a condition was evidence of a strong epileptoid tendency I did not doubt, and my forebodings of the ultimate result were fulfilled, for after the lapse of about two years he returned to me with no hyperæsthsia of the skin of his neck, but with occasional fully-developed epileptic paroxysms, for which he is now under treatment. Inquiry, however, showed that they were the result of late hours and indiscretions in diet, and that apparently they had no connection with the former series of attacks.

Relative to this subject of convulsion without loss of consciousness but appearing paroxysmally, I shall have some remarks to make in the next chapter, under the head of "Convulsive Tremor."

It has been urged by some writers that migraine is a modified epilepsy. Dr. Hughlings Jackson would certainly regard such cases as those of Sir John Herschel, the astronomer-royal, the late Sir C. Wheatstone, Dr. Hubert Airy, and, going farther back, those of Dr. Parry and Dr. Wollaston, as genuine epilepsy. Dr. Latham,[1] in his very instructive little book, from which I cite these examples, quotes as follows Sir John Herschel's account of the phenomena observed in his own case, in which there were present in his field of vision irregular fortification-like figures, the margins of which were colored:

"In one attack in myself, which occurred while I was conversing with an acquaintance, I soon became painfully sensible that I had not the usual command of speech; that my memory failed so much that I did not know what I had said or had attempted to say, and that I might be talking to another."

Dr. Airy, who has also described his own case, says:

"Sometimes the speech is affected, and the memory at the same time. On one occasion the mouth was seen to be drawn to one side."

---

[1] "On Nervous or Sick Headache," Cambridge (England), 1873, p. 10; also, *Philosophical Magazine*, vol. xxx., p. 21.

In a young female who came under Dr. Latham's observation, and who had colored spectra, there was a tingling of the arm and the side of the tongue, and on the same side with the spectra. Her sister and father were affected in precisely the same way. In another case the patient complained of a feeling of pinching and scratching on that side of the face corresponding with the glimmering.

In most of these cases these spectra and sensations were followed by headache of severe character, attended with nausea and vomiting.

But, notwithstanding the resemblance to epilepsy which all these phenomena of migraine suggest, Dr. Latham asserts that it differs widely from that terrible disorder in that it never threatens life, is never associated with unconsciousness, and that he has never known it to pass into epilepsy. On the contrary, with advancing age the attacks, as a rule, become much less frequent. They cease generally after fifty or sixty, and in women, not uncommonly, at the change of life.

Dr. Latham holds the view that migraine is an affection of the sympathetic system; that the ocular spectra are the result of an anæmic condition of the brain due to a tonic contraction of the arteries; and that the pain which subsequently appears is the result of arterial relaxation and consequent cerebral congestion.

In his most thorough and valuable work Dr. Liveing[1] discusses the whole subject of migraine in all its relations; and, while admitting with Marshall Hall, Sieveking and others that very intimate relations exist between sick-headache and epilepsy, and adducing several examples in which epilepsy has occurred to persons who were in previous years subject to the former affection, nevertheless regards such occurrences as quite exceptional, and as instances only of that occasional metamorphosis of neuroses so often witnessed.

That migraine is an affection of the vaso-motor system is rendered very probable by the observations of Möllendorff,[2] who reaches the conclusion that it is the consequence of arterial hyperæmia. He found that ophthalmoscopic examination of the eye of the affected side revealed the existence of dilatation of the arteria centralis retinæ as well as of the choroidal vessels and of a bright-scarlet color of the fundus, while on the other side the vessels were normal, and the fundus, of its usual dark-red color.

This theory is adopted by Dr. Bergen[3] in a recent elaborate paper. It is the very opposite to that proposed by Dr. Bois-Reymond, according to which migraine is due to a tetanic contraction of the cerebral arteries. Neither of these authors regards migraine as a form of epilepsy.

---

[1] "On Megrim, Sick-headache, and some Allied Disorders, a Contribution to the Pathology of Nerve-Storms," London, 1873.

[2] "Ueber Hemicrania," *Archiv. für pathologische Anatomie*, Band xl., p. 385.

[3] "On the Pathogenesis of Hemicrania," translation from the German by Dr. H. Gradle, in the *Chicago Journal of Nervous and Mental Diseases*, vol. i., 1874, p. 296.

My experience with sick-headache has been quite extensive. I have frequently witnessed cases in which there were chromatic ocular spectra such as those described by Latham, Sieveking, and others, but I have never perceived anything more in the most marked forms of the affection than a resemblance to some of the phenomena of the epileptic attack. One very noticeable difference is as regards the effect upon the mind. In epilepsy the slightest and most transient seizures generally impair, after a time, the mental faculties, especially the memory, while in migraine, no matter how severe or how frequent may be the attacks, the mind in all its parts retains its full vigor.

In an interesting lecture on the pathology of epilepsy, Dr. J. S. Jewell,[1] of Chicago, expresses the opinion that the phenomena of an attack are due in the first instance to a spasm of the cerebral vessels by which anæmia is produced, which in its turn causes unconsciousness, and that the relaxation of the spasm causes cerebral congestion, the immediate consequence of which is convulsion of the voluntary muscles. This congestion, he believes, is mainly seated in the medulla oblongata, or at least that it is the congestion of this part of the encephalic mass that produces the convulsive movements. The starting-point in the pathology of the disease would therefore be in the vaso-motor system.

This theory is similar to—in many respects identical with—that propounded in the former editions of this work and given at the beginning of the present remarks. It is very obvious to my mind that epilepsy is something more than the result of a discharging lesion of gray matter, and, as I have said, I cannot accept the view that all such discharges are epilepsy.

Treatment.—The treatment of epilepsy rests almost solely on experience. To attempt the consideration of all the means which have been employed would be a fruitless task, even though it were possible. I shall therefore content myself with detailing the measures which I have found most useful.

Among medical remedies the bromides stand preëminent, and should be thoroughly tried first in every case. The bromide of potassium, sodium, or calcium, may be used. Of these the bromide of sodium is the most advantageous in the majority of cases. Its taste—that of common salt—is not unpleasant, and it agrees better with the digestive system than the potassium compound. The bromide of calcium deranges the system still less, but its taste is not so pleasant, and it is much more expensive. Whichever one is preferred, the dose for an adult in ordinary cases and in the beginning of the treatment is fifteen grains three times a day in solution.

---

[1] "Pathology of Epilepsy," *Chicago Journal of Nervous and Mental Diseases*, vol ii, 1875, p. 1.

It must be clearly understood that the bromide, if successful in arresting the convulsions, must be taken for a long time, in order to increase the probability of a cure. I never discontinue it under two years, and sometimes persevere with it still longer, if in the mean time there have been attacks of vertigo, auræ, or other epileptoid manifestations.

After the initial doses have been given for about two months, if there are no symptoms indicating extreme bromism, or if there has been no paroxysm, I increase the doses by one-half. If there have been paroxysms in the mean time, I increase one-half after each paroxysm, until they are arrested, or until I am satisfied that the bromide is inefficacious or injurious. I have sometimes been compelled to carry it to the extent of nearly two hundred grains a day, and to continue it at that quantity for eight or ten days. When the system is thoroughly under the influence of the remedy and the convulsions have ceased, the doses may be reduced; but they should not be discontinued.

The bromides are less efficacious in the nocturnal variety of epileptic seizures, and in those which consist mainly of loss of consciousness, than in the diurnal and strongly convulsive kinds. In the former, sometimes, they increase the number and severity of the attacks, and in such cases should of course be at once discontinued.

A point connected with their action must not be overlooked, and that is, the cachexia which so generally attends their administration in large doses. In a memoir,[1] published over six years ago, and which has been cited in another connection, I brought forward several cases in which this cachexia had been produced. Greatly-increased experience has convinced me that, though in general it never causes any permanently ill effects, frequently great constitutional disturbance is induced. In three cases large carbuncles were caused, in a few I have been obliged to suspend for a time the administration of the medicine, and in two cases death resulted, in one from the patient taking larger doses than were prescribed, and continuing them while not under my immediate care, and in the other from the supervention of pneumonia while under the full influence of the remedy.

But, I am very sure that the bromic cachexia is favorable to the eradication of the epileptic tendency, and I therefore endeavor to produce it as soon as possible. It appears in many cases to alter the whole organism of the patient to such an extent as to leave him, when it disappears, with his nutritive processes and his proclivities so modified that epilepsy is no longer possible. The physician will require all his firmness and courage to persevere in those cases in which the bromism is extreme, but he should not yield unless the phenomena are so intense and the strength of the patient so greatly reduced as to excite his gravest apprehensions.

---

[1] "On some of the Effects of the Bromide of Potassium when administered in Large Doses," *Journal of Psychological Medicine*, January, 1869, p. 46.

The phenomena indicative of bromism will be given further on under the head of toxic affections of the nervous system. It may be, however, mentioned here that in the peculiar faculty possessed by the bromides of lessening the reflex excitability of the pharynx we have a ready means of ascertaining the extent to which the system is under the action of the remedy. If the handle of a spoon be pressed gently against the posterior wall of the pharynx of a healthy person, slight nausea and efforts to vomit are at once excited; but, if such a person be subsequently brought fully under the influence of any one of the bromides, the irritability of that part is destroyed, so that nausea or vomiting is no longer excited by pressure.

Herpin[1] several years ago called attention to the salts of zinc in the treatment of epilepsy. He preferred the oxide, and for a long time I made extensive use of this preparation in the treatment of the disease in question. Latterly, however, I have used the lactate, and still more recently the bromide, with very definitely beneficial results. It is best administered in the form of a sirup—my formula is: ℞. Zinci bromidi, ʒ j; syrupus simplicis, ℥ j. M. ft. sol.—which may be given in doses of ten drops gradually increased to thirty or more three times a day. It should be given largely diluted, as being the less apt to excite nausea.

In several cases the bromide of zinc has proved exceedingly efficacious thus far in arresting the paroxysms where other bromides had failed. Bromism is not an attendant on its administration, and yet it is quite probable that the bromine of the compound exercises considerable curative influence. I have given it as long a time as six months consecutively without producing cachexia, and to the extent in some cases of forty grains a day.

A troublesome feature which often attends the administration of the bromides—except the zinc compound—is the cutaneous eruption. Arsenic has been said to obviate the tendency to this complication, and to cure it where already present. In a few cases I have seen the use of the drug—four or five drops of Fowler's solution with each dose of the bromide—prove serviceable; but in the majority of cases it has appeared to be inefficacious. Owing to the supervention of carbuncles with a strong predisposition to gangrene of the skin, I have been obliged in several cases to discontinue the bromide of potassium. The calcium compound is, I think, not so liable as those of potassium, sodium, or ammonium, to cause this trouble.

In the nocturnal form of epilepsy strychnia is sometimes remarkably efficacious. It may be given in the beginning in the dose of the thirtieth of a grain three times a day gradually increased. A good formula for its administration is: ℞. Strychnia sulph., gr. ij; aqua dest., ℥ j. M. ft. sol. Dose, eight drops three times a day for the first two weeks, then nine drops for the next two weeks, increasing a drop every two weeks for a year, and perhaps longer.

---

[1] "Du pronostic et du traitement curatif de l'épilepsie," Paris, 1852.

Strychnia is also said to be useful in epilepsy of stomachal origin, that is, cases produced by gastric derangement.

The nitrite of amyl, first proposed and used in epilepsy by Dr. Weir Mitchell, is certainly beneficial in arresting the paroxysm, when there is an aura sufficiently pronounced and slow to give the patient the time to employ it. Five to ten drops may be inhaled from a handkerchief with safety, and generally with success. As there is generally not time to pour it out, this quantity should always be kept on the person in a glass-stoppered vial ready for use at a moment's notice. Dr. McBride, of this city, has had made little hollow thin glass beads containing the proper quantity of the nitrite of amyl, and when the patient experiences the warning, one of these is crushed in a handkerchief and the vapor inhaled through the mouth.

Dr. Crichton Browne[1] has not only used the nitrite of amyl in preventing individual paroxysms, but has given it with advantage with the view of breaking up the *status epilepticus*—a condition in which the fits succeed each other with scarcely an intermission, the patient being unconscious during such intervals as occur. The results of his experience are such as to convince him that it will be found invaluable in many cases, not only in postponing the paroxysm, but altogether preventing epileptic seizures.

It may be stated that the effect of the nitrite when inhaled is to accelerate the action of the heart, to make the face red, and to cause a feeling of fullness in the head, and a sensation as if pins and needles were sticking into the skin of the face, neck, and chest. These phenomena disappear in a few moments.

Within the last few years I have used the nitrite of amyl internally with, in some cases, decided benefit. It may be given in doses of from half a drop up to three or four, gradually increased, if necessary, and should be continued for a long time. A good formula is: ℞. Amyl nit., ♏ x; alcoholis, ♏ xc. M. ft. sol. Dose, from five to twenty or thirty drops a day. I usually begin with five drops on a lump of sugar, morning, afternoon, and bedtime, increasing the doses one drop every week so long as it continues to control the disease.

Several cases of epileptiform seizures clearly due to syphilitic infection have been under my charge, and have been treated with benefit by the bromides in conjunction with the iodide of potassium. In five of these, cures are known to have been effected.

As regards other medicinal remedies for epilepsy, I have but little to say. Belladonna has never in my hands produced the least good effect, neither has the nitrate of silver, nor indigo, nor cotyledon umbilicus, nor digitalis, nor any of the salts of copper. The same may be said of a hundred other substances less favorably known. Hydrate of chloral in three cases mitigated the frequency of the paroxysms, but only for a short time. Calabar bean was slightly beneficial in one case. Borax, after a very full trial, absolutely failed.

---

[1] "Nitrite of Amyl in Epilepsy," "West Riding Lunatic Asylum Medical Reports," vol. iii., 1873, p. 151.

But the whole treatment of epilepsy is not confined to drugs. Surgical and hygienic measures are often in the highest degree beneficial, and the latter should be brought into action in every case.

Of the surgical means the excision of any cicatrix which, by entangling a nerve, may be a source of reflex irritation, is occasionally a useful measure. This point has recently been brought forward in an interesting memoir by Dr. F. D. Lente,[1] in connection with cicatrices of the scalp, but the like reasoning and action are applicable to cicatrices existing in any other part of the body from which an aura appears to start.

In injuries of the skull, followed by epilepsy, trephining may be of great service. It has been aptly said that no blow upon the head is slight enough to be despised, and, so far as epilepsy is concerned, this is preëminently true. I have, during the past five years, trephined twenty-three times for epilepsy, which was apparently due to cranial injuries. In seven of these the fits ceased, and in two of the seven cases there was neither fracture nor depression. Of the remaining sixteen cases there was no cranial injury to be found in three; and in thirteen, though there was such injury, the operation proved unsuccessful, though beneficial results in lessening the frequency of the attacks were obtained in the majority. In one of them the fits did not recur for over a year.

The fact that in two of the successful cases no fracture or depression was found is a strong point in favor of Dr. Lente's view that epilepsy is sometimes the result of a cicatrix of the scalp, for, in both, the incisions in the scalp, as in all the others, were made so as to include the scalp-wound.

As the result of my experience, I am decidedly of the opinion that, in all cases of epilepsy in which there is injury of the skull or scalp, trephining or excision of the cicatrix should be performed, as may be proper.

In some cases counter-irritation to the nape of the neck is decidedly beneficial. It may consist either of a seton, which may be left in for several months, or the repeated application of the actual cautery. Counter-irritation is especially indicated in those cases in which the tongue is bitten, and instances in which internal remedies have failed till they were supplemented by this means are not uncommon.

---

[1] "Neuralgia and other Neuroses arising from Cicatrices of the Scalp, and their Surgical Treatment," "Transactions of the American Neurological Association," vol. i, New York, 1875, p. 157.

The hygienic management of the patient is important. A large portion of the day should be passed in the open air; bodily exercise should be regular, but not excessive; the food should be nutritious, but neither exciting nor indigestible. The importance of avoiding every alimentary substance, calculated to cause gastric or intestinal irritation, cannot be over-estimated. I have frequently seen paroxysms directly caused by nuts, dried fruits, pastry, heavy and badly-baked bread, excess in the use of alcoholic liquors, confectionery, and the like. And a diet consisting mainly of farinaceous substances is certainly preferable to one in which meat forms the larger part. I have in three cases effected entire cures by confining the patients for several months to a diet consisting at first of skim-milk, to which after a time a little bread was added. The bowels must be kept regular. Baths should be frequently taken, but should not be so cold as to cause severe shock or physical depression. Turkish baths, I am inclined to think, are useful in many cases, particularly in those occurring in persons of full and gross habit of body.

Overheated and ill-ventilated apartments should be avoided. The clothing should be warm in winter and cool in summer. The mind should not be overtasked, and the emotions must not be unduly excited.

Individual attacks may sometimes be prevented by other means than the nitrite of amyl. One gentleman under my charge assures me that he can often dissipate the premonitary symptoms, and thus stop the development of the paroxysm, by a strong exertion of the will. Another can arrest them sometimes by changing the position of his body. If standing, he lies down; if lying down, he rises suddenly and paces the room violently. Another stops them by putting salt in his mouth, and two can frequently prevent them by tightening straps which I have instructed them to keep constantly around the wrist. In all these cases there is an aura, and in the two latter it appears to start from the hand.

But, before resorting to any specific treatment for epilepsy, diligent search should be made for the cause, and this should be removed, if possible, without the least delay. Often an eccentric irritation, such as worms in the intestinal canal, implication of a nerve in an injury, disorders of menstruation, etc., can be discovered, without the removal of which a permanent cure is impossible. In several of the cases cited, success in the treatment was in a great measure due to acting on this principle.

The treatment during the paroxysm remains to be considered. It is simple, and, beyond a few obvious measures, consists in letting the patient alone. The head should be elevated, the collar and cravat loosened, a piece of soft wood put between the teeth so as to prevent injury to the tongue, and the patient so placed that he cannot fall or otherwise injure himself in his struggles. During the subsequent stupor he should be kept quiet. Bloodletting is never necessary, although it is recommended as proper in certain cases by Jaccoud.

CHAPTER III.

CONVULSIVE TREMOR.

UNDER the designation of convulsive tremor, I propose to include all those cases of non-rhythmical tremor or clonic convulsive movements which are unattended with loss of consciousness, but which, nevertheless, are paroxysmal in character.

As the affection has not yet found its way into the systematic treatises I shall, as in the matter of spinal irritation, devote a few words to its history, and, in so doing, shall draw largely from a paper of my own on the subject, published over eight years ago,[1] and from a clinical lecture[2] delivered to the class at the Bellevue Hospital Medical College in the winter of 1871–'72.

History and Symptoms.—In the year 1822 Dr. Pritchard,[3] under the name of convulsive tremor, gave an account of two cases, presenting somewhat similar features to the one before us. His attention was first directed to the subject by noticing that, in some epileptic patients who had come under his observation, fits of tremor occurred in the intervals between the paroxysms and even appeared to take the place of the ordinary seizure. He then noticed several cases in which there were no epileptic attacks, but in which there were violent clonic spasms of the muscles, accompanied with severe pain in the head and profuse perspiration. Dr. Pritchard states that, previous to his observations, the affection had not attracted much attention; but he cites a case from Tulpius of a young unmarried woman, of a pale complexion and phlegmatic temperament, who was afflicted during three years with what was called the shaking-palsy, which did not affect her constantly, but came on in periodical fits; each paroxysm lasted nearly two hours, and was accompanied by hoarseness and loss of voice, the consciousness being unimpaired.

He also refers to other cases quoted by Sauvages from Bonetus, in which the symptoms were very similar, consisting of convulsive tremor, attended with headache and vertigo. This disorder was fatal in a few days, and after death a parasite was found in the brain. In this connection it is interesting to recall the fact that the sheep is subject to a somewhat similar train of symptoms, due to the presence of an entozoön in the brain.

---

[1] "On Convulsive Tremor," *New York Medical Journal*, June, 1867, p. 185.

[2] "Clinical Lectures on Diseases of the Nervous System," New York, 1874, p. 164.

[3] "A Treatise on Diseases of the Nervous System," London, 1822, p. 393.

Dr. Pritchard then relates his own cases, of which the following account is an abstract:

John Pugh, a carpenter, of meagre habit, low stature, and dark hair, aged fifty, was admitted into St. Peter's Hospital March 1, 1820. About a month previously he had suffered from tonsillitis and subsequently had some difficulty of breathing, which was supposed to be asthmatic. He had complained of headache for some time. On the 23d of February he was attacked with a violent tremor, which continued for two or three hours, and then went off after he had taken an emetic. It recurred on the following day at the same time, and on every succeeding day about the same hour. At the time of his admission he was laboring under a paroxysm.

On first looking at the man, Dr. Pritchard supposed him to be in the cold stage of intermittent fever, but on closer and more careful examination his affection was seen to be very different. All the muscles of the upper extremities, including those connected with the ribs, clavicle, and scapula, were constantly agitated by a convulsive movement which was almost entirely confined to them. The lower extremities were quite free from disorder. The man was perfectly conscious, and able to answer any question distinctly. His pulse was quick and apparently irregular. Owing to the constant agitation of the tendons it was impossible to arrive at certainty on this latter point. The skin was warm, and there was no sensation of chilliness. The upper part of the body was in a state of profuse perspiration. He complained of vertigo and headache.

Bloodletting was ordered; a large orifice was made, and the blood allowed to flow till thirty-eight ounces had escaped, when syncope ensued. When half the above quantity had passed, the tremor became more general and severe. The gluteal muscles were so greatly convulsed that by their action the patient was thrown up from his seat with the motion of a man sitting on a trotting horse. When he became sick and faint, the arm was tied up and he was laid upon a bed. The tremor immediately ceased, except some slight and partial quivering.

He was then strongly purged, and this operation was continued every night. On the 5th, at 11 A. M., the tremor returned. Cold effusion was directed; as soon as the cold water was thrown over him the tremor ceased.

On the 9th there had been no return of the tremor. Calomel and sulphate of magnesia were now prescribed and on the 11th the tremor returned, lasting, however, but about twenty minutes. From this time he was free from the affection, but, as might have been expected, when the character of the treatment is considered, he fell into a state of debility. There were loss of appetite, cough, expectoration, and inflammation of the vein, ensuing from the bleeding.

In the next case the paroxysms of tremor were the most remarkable feature, but there were also stupor and delirium.

John Jones, a seafaring man, aged twenty-five, was brought to the hospital March 11, 1819, under a warrant of lunacy; was in the habit of drinking spirituous liquors. Three weeks previously he was seized with rigors, attended with coldness, and followed by heat, headache, and wandering pains in the limbs. The symptoms ushered in a state of stupor and delirium, during which his countenance became distorted, the eyes rolled, the muscles of the face were slightly convulsed, and the body was generally agitated. After a time all these symptoms subsided and he became perfectly rational, but seemed a little stupid, as if roused from a sleep. The paroxysms returned at uncertain intervals and with the same succession of symptoms. He was bled and purged, and finally brought to the hospital.

On admission he was in a state of delirium. He rolled his head about and was in constant motion. The temporal arteries beat rapidly and forcibly; the scalp was hot, the feet cold; face flushed and tongue a little furred.

His head was shaved and covered with cold wet pads, his feet were immersed in hot water, twenty leeches were then applied to the head, and calomel and tartar-emetic with cathartic draught administered.

The next day he was rational, but, as he complained of pain in the head and in the region of the liver, and as his pulse was 130, full and jerking, he was bled to the extent of eight ounces; syncope followed. Twelve leeches and a blister were then applied to the right hypochondriac region, and calomel, cathartic draught, and low diet ordered.

Notwithstanding the treatment, he continued to survive, and in the evening had two returns of the tremor followed by the usual symptoms.

On the 14th had several paroxysms, and was again freely purged; was occasionally bled from the temporal artery, and often leeched freely. Nitrate of silver was subsequently administered, and on the 23d of June he was discharged cured.

Dr. Pritchard states that he met with two other instances of paroxysms of tremor unaccompanied with spasm, and occurring in persons who had suffered from an attack of paralysis.

Evidently Dr. Pritchard has embraced two or three separate affections under the designation of convulsive tremor. The first case I have quoted from him appears to be a distinct and not previously-described disorder; the second case was probably one of cerebral congestion or aborted epilepsy; and those which he states he had seen as the sequence of paralysis were doubtless to be classed under some one or other of the forms of sclerosis of the brain and spinal cord. The first case alone is to be regarded as one of convulsive tremor, as described in this chapter.

In his very excellent treatise on the shaking-palsy, Parkinson,[1] in calling attention to the fact that several diseases characterized by tremor have been confounded with paralysis agitans, quotes the following case from Dr. Kirkland :

"Mary Ford, of a sanguineous and robust constitution, had an involuntary motion of her right arm, occasioned by a fright, which first brought on convulsion-fits and most excruciating pain in the stomach, which vanished on a sudden, and her right arm was instantaneously flung into an involuntary and perpetual motion like the swing of a pendulum, raising the hand at every vibration higher than the head ; but, if by any means whatever it was stopped, the pain in her stomach came on again, and convulsion-fits were the certain consequence, which went off when the vibration of her hand returned."

Parkinson also quotes another case from the same source, resulting apparently from worms, and which is thus described :

"A poor boy, about twelve or thirteen years of age, was seized with a shaking-palsy. His legs became useless, and, together with his head and hands, were in continual agitation ; after many weeks' trial of various remedies, my assistance was desired. His bowels being cleared, I ordered him a grain of opium a day in the gum-pill ; and in three or four days the shaking had nearly left him. By pursuing this plan, the medicine proving a vermifuge, he could soon walk, and was restored to perfect health.

Toulmouche,[2] in a paper which is very suggestive in the light of recent contributions to neurological pathology, cites a case which was evidently one of convulsive tremor :

"A woman, whose respiration was convulsive, presented from time to time the following condition : Her nostrils were strongly dilated, the angles of the mouth drawn down, the shoulders and chest spasmodically elevated, the inspiration strong and deep, the sterno-cleido-mastoid muscles were powerfully contracted. During these paroxysms, which lasted several minutes, the patient was deprived of the faculty of speech and threatened with suffocation. Nevertheless, she could, if so directed, move the head, the shoulders, and the muscles of the face, although the spasm continued. . . . In another case the affection was almost entirely confined to the sterno-cleido-mastoid muscle. The patient could turn the head in either direction, but gradually it moved from right to left, without her ability to control its action, so that the right ear almost rested upon the sternum. The other muscles of the shoulder contracted at the same time. He likewise reports another case in which the head was almost continually in motion, the patient executing twenty-two rotations in a minute; the movement was due to the alternate contraction of the sterno-cleido-mastoid and splenius muscles of each side; respiration was not obstructed. The movements diminished and finally ceased after two or three attacks of hæmoptysis.

---

[1] "Essay on the Shaking-Palsy," London, 1817, p. 29.

[2] "Observations de quelques fonctions involuntaires des appareils de la locomotion, et de la préhension," "Mémoires de l'académie royale de médecine," tome deuxième, Paris, 1833.

"The conclusions which the author draws from his own cases, and those which he cites from other authorities, are mainly interesting in relation to his theory of the pathology. They are—

"1. That there exist, for the movements of the different groups of muscles, different central motor forces.

"2. That the cerebellum only presides over the coördination of those complex movements which are necessary to the different acts of standing and locomotion, and not at all over those that regulate the more simple movements of the trunk and the members.

"3. That this nerve-centre supplies to vertebrate animals the power to maintain themselves in equilibrium and to exercise locomotion.

"4. That, if, in the species of neurosis I have described, the sensation and the intellectual faculties experience no change, this fact is due to the circumstance that the lesions of the cerebellum have not yet involved the tubercula quadrigemina. That these last-named organs are in a state of dependence upon the brain; since in the normal state animals move through the impulsion of various motives of which the brain is the seat.

"5. That finally a number of affections called nervous, consisting in the most erratic derangements of the muscular functions, such as an irresistible tendency to go backward or to advance without rational motive, to leap, to perform other disorderly movements, constitute only a species of insanity or aberration of the locomotor functions depending on an affection either organic or functional of the cerebellum."

I have quoted the conclusions of Toulmouche in full more as evidence of the fact that he was disposed to locate the seat of these troubles in the cerebellum, than as intending to endorse his collateral hypotheses. At one time I also held the opinion that the seat of convulsive tremor was in the cerebellum, but I have for some time had a different idea on the subject.

Up to the publication of my own paper, in 1867, there had been no attempt made to define accurately the features of the disease under notice. My description of the affection was based upon three cases. They were as follows:

Case I.—J. S., a gentleman, aged thirty-five, single, and engaged in mercantile pursuits, consulted me on March, 14, 1867, for an affection which, as he said, "was driving him mad." Ordinarily he had nothing to complain of on the score of health. His appetite was good, and all his functions were performed with regularity; but two or three times during the course of the day he would be seized with severe and uncontrollable muscular tremor, involving his head and all the muscles of the trunk and arms. At the same time there would be headache and an intense feeling of anxiety. There was no loss of consciousness, not even for an instant, or inability to walk or to direct any muscle, and no confusion of thought. After the paroxysm had lasted fifteen or twenty minutes it gradually passed off, leaving him in a profuse perspiration.

While he was sitting in my library an attack came on. He was seized with as much suddenness as though he were struck with an epileptic fit. His head shook violently, the muscles of his face were convulsed, his arms and hands trembled, and his gluteal muscles contracted so powerfully as to cause him to move convulsively up and down on his chair. His lower extremities were altogether free from spasm or convulsion. Upon putting my hand on his wrist, I found that every tendon was in action, and in the arm, hand, neck, and face, the vibration of the muscular fibres could be distinctly seen and felt. I thought the action was greater on the left than on the right side.

The thermometer applied to the axilla marked 101° Fahr., and the æsthesiometer showed an increased sensibility of the skin of the face, neck, hands, and all the upper parts of the body I examined. The respiration was quickened, and the pulse was increased from 80 to 95 per minute.

During the continuance of the paroxysm he conversed rationally but with some difficulty, owing to the action of the muscles of the neck, mouth, and chest. The pupils contracted briskly under the influence of light, and dilated when it was shut off. Several times he rose from his chair and paced the room; his movements were perfectly well made. There was a little headache, confined to the occipital region, and a slight but persistent vertigo.

I desired him to perform a few movements with his hands, such as buttoning his waistcoat. He had no great difficulty in carrying his hands to the buttons, but it was impossible for him to seize them, and the more his efforts were directed to this end the more difficult it was for him to accomplish it. The trouble was not in loss of strength, for, when I asked him to grasp my hand, he did it with great force, but the tremor was so constant that he could not keep the ends of his fingers at any one point.

After the paroxysm had lasted about fifteen minutes it began to subside, and in ten minutes more had entirely passed away. The thermometer in the axilla now marked but 98° Fahr., and the hyperæsthesia had entirely disappeared, leaving the sensibility of the skin natural. The respiration and pulse became normal in frequency.

Upon questioning this gentleman, I ascertained that he had indulged to excess in venereal pleasures, and that the first attack of tremor had begun during sexual intercourse. He said that, just as the orgasm was approaching its height, he had experienced a severe pain in the back of his head, accompanied with tremor. That, notwithstanding, he had completed the act, but felt very greatly debilitated after it; the tremor continued for a few minutes, and then passed off. This was about four months before I saw him. Since the beginning of his disease he had entirely abstained from all sexual indulgence, but his tremors had not left him for a night or day. In consequence he was low-spirited, and apprehensive of losing his reason.

Case II.—The second case was that of a young lady, aged twenty-one, who was sent to me March 21, 1867, by Dr. C. F. Taylor, to whom she had gone to be treated for lateral curvature of the spine. In addition to this trouble she had for four years been afflicted with a disorder, certainly very singular in its characteristics, and for which she had been treated by many physicians of many systems of practice. The chief and most distressing feature was a spasmodic action of the diaphragm coming on every ten or fifteen minutes, producing convulsive respiration, a feeling of impending suffocation, and great mental anxiety. The paroxysms lasted four or five minutes, and then passed off with a long, deep-drawn sigh. None of the respiratory muscles but the diaphragm were convulsed. By placing the hands over the abdomen this muscle could be distinctly felt in a state of rapid and irregular action. In the intervals of the diaphragmatic paroxysms, there were frequent tremors of the arms, legs, and head. There was almost constant headache extending across the crown to the cerebellar region. There was no fever or increased temperature, but great hyperæsthesia of the whole surface of the body. The menstrual function was normal in every respect, and there was no evidence of hysteria. Her appetite was bad, and what she did eat was not of a very nutritious character. Occasionally she was subject to fits of great mental and muscular excitement, during which she fought and bit all who came near her, but there was no mental aberration. She had never been subject to intermittent fever.

In this case the convulsive tremor, though more prominently manifested in the diaphragm, was not confined to this muscle, for, as I have stated, when it was quiet the muscles of other parts of the body were in irregular but rapid action. There was not the entire cessation of tremor as exhibited in the first case, and the paroxysms were much less uniform and much less extensive in their character. In both cases the tremor was absent during sleep.

Case III.—In a third case the patient was a young man aged twenty-five, and a clerk by occupation. He came under my care April 2, 1867, to be treated for obstinate headaches, with which he had been affected for several years. On an average he had an attack twice a week of so severe a character as to unfit him for all occupation and to confine him to bed. The pain was limited to the back part of the head, and was exceedingly sharp and lancinating; vertigo and an indescribable twisting sensation within the cranium accompanied the attack. In addition there was convulsive tremor of the muscles of the head, face, and neck, occurring in paroxysms at intervals while the headache lasted. There was no loss of consciousness and no confusion of thought. There were, however, great physical prostration, and an indisposition to make any mental exertion.

In his youth he had, as he informed me, practised masturbation to excess, and since attaining to manhood had indulged freely with women. He was also addicted to the abuse of alcoholic liquors. He was thin, pale, and of deficient vital power. His digestive system was deranged, his appetite bad, his pulse weak and frequent. There was no disease of the lungs or heart. He had had gonorrhœa and stricture, but had never contracted a chancre.

He had been under the charge of several physicians, but had never been able to subject himself to the regimen and restrictions in his habits of life which they recommended. Latterly he had undertaken to treat himself, and had done so mainly by inhalation of chloroform.

This patient would not abstain from debauchery of all kinds, and I dismissed him.

Case IV.—A fourth case formed the subject of a clinical lecture which I delivered three years since before the class of the Bellevue Hospital Medical College. The patient, a young man, aged about twenty-one, was well nourished, of general healthy appearance, and by occupation a farmer.

At periods varying from a few weeks to several months, he was subject to violent convulsive movements in almost all the muscles of the body, and unattended, except in one instance, by loss of consciousness. The paroxysms lasted several hours, and during their continuance the patient, owing to the violent jactitations into which his limbs were thrown, was totally unable to execute voluntary movements. He was even unable to stand without support, and could not guide either his hands or feet. The muscles of speech were likewise affected, and he was consequently unable to articulate distinctly the words he might attempt to utter. While all this was going on, his body was bathed in cold perspiration, and the circulation was accelerated. The respiration was increased in frequency, and there was well-marked and persistent pain in the back of the head and nape of the neck. He was very positive that, except in the one instance to which reference has been made, he had never lost consciousness during a paroxysm, but had always been possessed of his full reasoning faculties.

On the occasion of loss of consciousness the paroxysm had lasted several hours; he was in consequence very much exhausted, and therefore he may have been suffering from simple syncope, still it is possible the attack in question was epileptic. When he came under my notice, he had been affected for about six years.

As he described his paroxysms, the muscles were affected very much as are those of a person suffering from chorea of very violent character.

CASE V.—A fifth case was that of a man thirty years of age, who, in November, 1875, came to my clinic for diseases of the nervous system at the University Medical College. At intervals through the day, as often as twenty or more times, he was seized with violent convulsive movements, tremulous in character, and mainly confined to the muscles of the trunk, neck, and upper extremities. As in the other cases, there was no loss of consciousness, nor was there any other mental disturbance. He had no power of control over these paroxysms and no warning of their approach. They were unattended with disturbances of the respiration, circulation, or sensibility. The duration rarely exceeded ten seconds, and was generally shorter than this. It was impossible to say where the convulsive movements originated. They came more as an explosion than as a gradually-developed action.

While the convulsion was at its height, he could always cut it short and prevent others for a time by smoking a pipe of tobacco, the requisites for which he kept constantly ready. He had been affected for seven years, but had in that time experienced an intermission of about six months. He had never had a paroxysm while asleep.

CASE VI.—This case was that of a lady from Ohio, who consulted me for paroxysms of convulsive tremor, coming on several times in the course of the week, and involving the upper and lower extremities and neck. There were also marked disturbances of the respiration and circulation, and pain in the nape of the neck. The movements consisted of rapid but limited flexions and extensions of the limbs and rotatory movements of the head. The duration of a paroxysm was rarely less than ten minutes, and sometimes was several hours. There was no mental disturbance or impairment of consciousness. The disease had existed for several years, and had proved unamenable to all medical treatment. After each seizure there was a very intense feeling of fatigue, but no tendency to sleep or stupor. No paroxysm had ever occurred during sleep. The general health was excellent, and the mind was active and strong.

Several other cases similar in general features to the foregoing have been under my charge.

From this history and description it will be seen that convulsive tremor is an affection characterized by paroxysms of clonic convulsions affecting the voluntary muscles and unattended by loss of consciousness or by mental aberration, though sometimes there is emotional disturbance. Vertigo and pain in the head are also occasional accompaniments.

Causes.—Nothing very definite is known relative to the etiology of the disease. In one of my cases it began during sexual intercourse;

in another (Case V.) it ensued immediately after a sunstroke, the first paroxysm occurring while the patient was still in a comatose condition; in another (Case III.), sexual and alcoholic excesses appeared to be the cause. In none of the others could any approach to a relation of cause and effect be established.

Diagnosis.—From epilepsy, convulsive tremor is distinguished by the absence of loss of consciousness. Many of the cases which Dr. Hughlings Jackson considers epileptic are, in my opinion, more properly embraced under the present category. From chorea it differs in the facts that the muscular action is paroxysmal and not continuous, and that the movements are different in character, those of convulsive tremor being rapid and tremulous, while those of chorea are slower and more systematic. The paroxysmal nature of the actions serves to distinguish it from athetosis, multiple cerebral sclerosis, multiple cerebro-spinal sclerosis, and paralysis agitans. From hysteria it is in uncomplicated cases diagnosticated by the absence of other symptoms of the hysterical condition, by the fact that the convulsions are not marked by tonic spasms, and the circumstance that they have for each individual case a definite character.

Prognosis.—The prognosis is generally favorable, the disease, in my experience, being quite amenable to medical treatment. All the cases under my care recovered except one in which the patient refused to submit to proper hygienic restraints, and in whom treatment was not therefore systematically pursued.

Morbid Anatomy and Pathology.—In former papers I have stated my belief that convulsive tremor was an affection of the cerebellum, but in the light of the investigations of Fritsch and Hitzig, Nothnagel, Ferrier, and Bartholow, I am now disposed to consider it due to irritation of nerve-centres in the cortical substance of the cerebrum, conjoined with a hyperæsthetic condition of the medulla oblongata and upper part of the spinal cord. And I am the more confirmed in this opinion by some recent experiments by which I have ascertained that a very similar disorder can be induced in dogs by the faradization of the parts mentioned.

Ferrier[1] produced epileptiform convulsions in rabbits by faradizing the greater part of a hemisphere. In one of my own experiments I exposed both hemispheres and applied to each a piece of chamois-skin thoroughly moistened with water, and cut to fit the surface. The electrodes—metallic buttons—were, then placed one on each piece of chamois-skin, and moved lightly over the surfaces for a few seconds. The animal was then allowed to emerge from the anæsthetic condition, and immediately general convulsive movements ensued without loss of consciousness. The result was, therefore, similar to that obtained by

---

[1] "Experimental Researches in Cerebral Physiology and Pathology," "West Riding Lunatic Asylum Medical Reports," vol. iii., 1873, p. 30.

Ferrier, but so far as I can judge the convulsive movements were more general, and there was no pleurothotonos as in his cases. The paroxysm lasted about ten seconds, and was repeated, though not to the same degree of intensity, after an interval of three minutes. During the next half-hour there were repeated localized convulsive movements in various parts of the body.

In another dog I exposed both hemispheres, and also the upper part of the spinal cord, as far down as the fourth cervical vertebra. A piece of wet chamois-skin was then laid upon the brain, and one electrode—a thin plate of copper—placed in contact with it, while the other—a thin copper wire doubled upon itself—was moved up and down upon the exposed spinal cord. During this operation the animal was in a state of general convulsion, the respiratory muscles, especially the diaphragm, being involved. The current was passed in this manner for ten seconds. The animal was then allowed to recover consciousness. As soon as the effects of ether had measurably passed off, convulsive movements ensued throughout the body, the diaphragm being markedly affected with the other respiratory muscles, and the heart beating with great irregularity, both in regard to force and rhythm.

In these experiments a Gaiffe's faradaic machine was employed, and the current was so feeble as barely to move the hammer and to be felt when the electrodes were applied to the tongue.

I think with Dr. Hughlings Jackson that such convulsive movements are the result of "discharging lesions" of nerve-centres. The case of the patient to which I have referred under the head of epilepsy, in whom there was convulsive tremor of one side of the neck and face, induced by irritation applied to the skin of that side, shows, as well as others on record, that such instances may be developed into epilepsy under adequate circumstances, but, as there said, I cannot regard them as primarily epileptic.

In another case—that of a young lady who has come under my care since the chapter on epilepsy was written, who is very excitable, has had two choreic periods, and once, certainly, an epileptic seizure—there are daily several attacks of convulsive tremor, in which the action starts from the right side of the neck, gradually invades the right side of the face, and eventually the muscles of the corresponding upper extremity. There is not for a moment the slightest impairment of consciousness. The face, however, is at first deathly pale, but soon becomes flushed. There is no stupor, no mental confusion before, during, or after the attack. She laughs and talks during its continuance, and has a perfect recollection of every thing that takes place during the paroxysm. That such a case is very near to epilepsy is undoubted, but then congestion is very near to inflammation, and may exist for years without advancing to full development.

There are certain morbid conditions usually classed as choreic, which have more affinity with convulsive tremor than with chorea, though, perhaps, they are, with even greater propriety, placed under the head of hysteria. These are the turnings, salaam-convulsions, jumpings, etc. It is quite probable that the lesion causing those disorders is similar to that producing convulsive tremor.

The morbid anatomy of the affection under notice is entirely a matter of supposition, and indeed there are not many data for forming an opinion relative to the essential nature of the structural alteration. So far as we can judge from a consideration of the phenomena, the seat is in the cortical substance of the brain, and in the medulla oblongata and upper part of the spinal cord. The disturbances of the respiration and circulation point to these latter organs as a part of the anatomical substratum.

In those cases in which there are spasms localized in various parts of the face, neck, or extremities, it is probable that the lesion exists entirely in a limited part of the cortical substance constituting the motor centre for the region involved.

Treatment.—In the first cases that came under my observation, I employed counter-irritation in the form of a seton inserted into the nape of the neck, large doses of the bromide of potassium, and the primary galvanic current. Iron and quinine were given in two cases to relieve the general anæmic condition which existed. These measures were entirely successful, except in the third case, in which the bromide of potassium produced no perceptible effect. The tincture of hyoscyamus was substituted for it with good results, but all treatment was subsequently abandoned as stated.

In the fourth case the patient was treated with gradually-increasing doses of strychnia, with the effect of causing a complete cure. A solution of the sulphate of strychnia, consisting of two grains to the ounce of water, was administered in doses of ten drops three times a day, the doses being increased by one drop every day, till the physiological effects of the drug were obtained. A return to the original dose of ten drops was then directed, and an increase as before. From thirty to thirty-five drops were generally necessary to cause slight rigidity of the muscles of the legs and neck. The patient continued treatment for several months, and had no further spasms.

In the fifth, sixth, and other cases, I have relied for internal treatment entirely on the bromide of zinc given in solution in gradually-increasing doses. In all of these the result has been entirely satisfactory. In the fifth case, no paroxysm ensued after the first day of treatment. Four weeks afterward, the patient presented himself at my clinic, and announced the complete cessation of all convulsive movements, and that he had resumed his work, which had been interrupted for several years.

In the sixth case I administered the zinc, and in addition applied the actual cautery repeatedly to the nape of the neck. Only one paroxysm occurred after the treatment was begun, and that was induced by the excitement and irritation caused by the primary galvanic current applied to the spine. The patient, two months afterward, remained entirely well, though still continuing to take the zinc.

In all the other cases, five in number, the bromide of zinc has sufficed to effect the cure.

I have uniformly given it in solution, either in water or simple syrup, in the proportion of one drachm to the ounce. Of this mixture, ten drops were given three times a day for the first two weeks, then fifteen drops three times a day for the next fortnight, and so on, increasing five drops for the doses of each subsequent two weeks. This course has been continued for from three to six months, and then the doses are gradually reduced, except in Cases V. and VI., in which I shall continue them for a much longer period, and in two others which have been but for a short time under treatment.

CHAPTER IV.

CHOREA.

ALTHOUGH it is quite certain that several distinct affections are included under the term "chorea," these are analogous to each other, and, as we know little about the essential anatomical features of these disorders, and as they are allied by their symptoms, it will be advisable, for the present, to consider them together.

Symptoms.—Even in simple, typical, and uncomplicated cases of chorea, the symptoms exhibit great variety. They are connected mainly with the mind, with motility, and with sensibility, though, at the same time, the functions of organic life are generally more or less deranged.

Among the earliest symptoms of chorea are those referable to disordered brain-action. The character and disposition of the patient undergo a marked change, and there is, besides, from the first, a very decided impairment of mental vigor. The emotions are easily excited, and the temper becomes fretful and irritable. Hallucinations are not uncommon, and these are generally connected either with the sight or hearing. Sometimes both these senses are involved.

The sleep is generally disturbed by disagreeable dreams, sometimes reaching to the intensity of nightmare, and these are so vivid that the patient often considers them realities.

In a few cases there is decided mania, but this is not of a very aggravated form, and is of temporary duration. Three such instances have recently been under my care, all occurring in young girls of about the age of puberty, and exhibiting in all other respects the typical characteristics of chorea.

In two cases under my observation, the first notable event in the course of the disease was an epileptic paroxysm, which, however, was not repeated in either case.

The most prominent symptoms of the disease are, in the great majority of cases, exhibited in the irregular and disorderly muscular contractions which make their appearance at a very early period, and which have given it a name in nearly every language of the civilized world. Thus, we have the terms chorea (*χορεία*, a dance), St. Vitus's dance, St. Guy's dance, etc.

In the beginning the foot of one side drags a little, and soon afterward the corresponding upper extremity becomes affected with the choreic movements. These are manifested in the fingers, in the flexion, extension, and rotation of the wrist, and in the movements of the elbow and shoulder. No matter where the hand be placed, it cannot be kept steady, but it and the whole extremity are in a constant state of agitation. Before long the muscles of the neck and face participate, the head is jerked from side to side, and a continual series of grimaces is the result of the actions in the facial muscles.

In some cases the involuntary movements are confined to one lateral half of the body, constituting the form known as hemichorea. This is the case in about one-fourth of the instances. Thus, of two hundred and thirty-five cases cited by Sée,[1] the phenomena in sixty-four were limited to one side. This limitation has not, as was formerly supposed, any relation with hemiplegia, but is solely the result of the suspension of the progress of the disease.

At first the movements are moderate, but they go on, becoming more and more severe, until, in extreme cases, the condition of the patient becomes exceedingly pitiable. The arms, the legs, the face, and head, are in almost constant action. Every attempt to perform a voluntary movement excites still more the disorderly actions, and thus the patient is unable to feed or dress himself, and sometimes even walking becomes impossible.

In one type of cases the convulsive movements come on paroxysmally, and are often of the most astonishing character. The patient is, perhaps, lying quietly on the bed, when suddenly the head is thrown backward, the limbs set in involuntary motion, and the muscles of the trunk contract so violently as to throw the sufferer forcibly to the floor. Again, a series of gyratory motions ensues, and the patient turns round on one foot until complete exhaustion follows; or there may be leaps and contortions of various kinds. Sometimes the movements are rhythmical. A lady, who a short time since was under my charge, was suddenly seized with an irresistible impulse to bend the left elbow. The arm continued in motion for half an hour, and then the right arm began a like movement. In a few minutes the head began to nod, then the left knee was alternately flexed and extended, and finally the right knee became similarly affected. For over an hour these movements continued, and then a regular alternation ensued—first the left arm, then the right, then the head, next the left leg, and finally the right leg. These actions were perfectly timed, and were all performed in exactly ten seconds, as I ascertained by determinations made on several occasions. As she sat in a chair, or lay on a bed, she was a curious sight. Though she was good-tempered with it all, her emotional system was in a state of great exaltation. She recovered in a few weeks.

---

[1] "De la chorée et des affections nerveuses en général, avec leurs rapports avec les diathèses, et principalement avec le rheumatisme," "Mém. de l'académie de médecine," 1850, tome xiv., p. 343, *et seq.*

In another case a lady from New Jersey was affected in a still more extraordinary manner. While sitting sewing one day, after having been greatly fatigued the previous night, her leg began to tremble violently. In a few minutes the arm of the same side became involved, and very soon the other limbs and the head were affected. She was now in a state of general tremor, and, on attempting to rise, fell to the floor. She was then seized with another kind of movement. Her legs were drawn up forcibly, and then suddenly extended, and this with inconceivable rapidity. She was placed on a bed, but was unable to stay there unless held by several persons, so strong were the contractions which took place. On one occasion she was thrown over five feet, her body coming to the floor with great violence.

The following day a fresh series of phenomena ensued. She began to turn somersaults, and continued these actions for several hours without appearing to be greatly exhausted. Then she jumped suddenly to her feet, and rushed round in a circle with such swiftness that she could not direct her steps, and she several times knocked her body with great force against the walls and furniture. Then she danced for several hours, and toward evening became tolerably quiet, though there was still involuntary twitching of nearly all the muscles. In all the various movements she went through, every attempt to hold her only made her worse, and she begged that she might be let alone, as the effort to control her by physical force made her head swim, and gave her a severe headache. At night the paroxysms ceased, but they were renewed as soon as she awoke in the morning, and continued with but little intermission, and in every possible form, till she went to sleep.

On the third day I visited her, and found her in the midst of a series of movements such as I have described. Her pulse was irregular, her respiration hurried, and her countenance evinced great anxiety. There was no evidence of any hysterical complication.

I at once proceeded to administer chloroform by inhalation, and in a few moments she was completely under its influence. The paroxysms ceased soon after the inhalation was begun. I kept her in a state of anæsthesia for half an hour. When she recovered consciousness she was perfectly composed, and remained so all the rest of that day. I left directions that the inhalation was to be repeated if there should be any returns of the choreic paroxysms, but there were none. She slept well all night, and the following morning was quiet till about eleven o'clock, when a slight tremor began, which was at once quieted by the chloroform. I saw her again that day, and began a treatment consisting mainly of strychnia in gradually-increasing doses, and renewed my directions in regard to the chloroform. After this she had a few attempts at paroxysms, but they were always stopped by the inhalation of the chloroform, and in a few weeks she was well.

Chorea of rhythmical or uniform character has often prevailed epidemically. The most authentic recorded visitation of the kind was one which occurred at Aix-la-Chapelle in 1374. This was in the form of a dancing mania, and is fully described by Hecker[1] under the name of St. John's dance. The men and women subject to it met in the streets and churches, where "they formed circles hand-in-hand, and, appearing to have lost all control over their senses, continued dancing, regardless of the by-standers, for hours together in wild delirium, until at length they fell to the ground in a state of exhaustion. They then complained of extreme oppression, and groaned as if in the agonies of death, until they were swathed in cloths bound tightly around their waists, upon which they again recovered, and remained free from complaint until the next attack. This practice of swathing was resorted to on account of the tympany which followed these spasmodic ravings, but the by-standers frequently relieved patients in a less artificial manner, by thumping and trampling upon the parts affected. While dancing they neither saw nor heard, being insensible to external impressions through the senses, but were haunted by visions—their fancies conjuring up spirits, whose names they shrieked out; and some of them afterward asserted that they felt as if they had been immersed in a stream of blood, which obliged them to leap so high. Others, during the paroxysm, saw the heavens open and the Saviour enthroned with the Virgin Mary, according as the religious notions of the age were strangely and variously reflected in their imaginations.

In the most fully-developed and best-marked instances of the disease, it was often ushered in by an attack of epileptic convulsions. Such were probably cases of hystero-epilepsy, an affection to be presently considered at greater length.

The affection spread like wild-fire—being fed by that principle of imitation which appears to be so powerful an influence in causing the propagation of this and analogous disorders of the nervous system. Those affected were generally regarded as being possessed by evil demons, and consequently only to be cured by the exorcisms of the clergy.

---

[1] "Epidemics of the Middle Ages," "Sydenham Society Translation," 1844, p 87.

In 1418 it broke out in Strasbourg, and there received the name of St. Vitus's dance, from the fact that the most efficacious means of cure was thought to consist in the intercession of this saint.

Similar attacks of dancing mania had occurred before that of St. John, but the details are more or less obscure, and several have occurred since. Among these latter must be placed the tarentism which overran Italy, and various more restricted epidemics of like disorders. In our own country we have had the Jumpers, and we still have the Shakers. In addition to these are many of the manifestations of witchcraft, which were choreic, and of which this country has had its full share, and of spiritualism, which it enjoys the doubtful honor of having initiated.[1]

In chorea, even of the ordinary simple kind, the speech is imperfect, owing to the incoördination of the muscles directly concerned in articulation, and those which effect respiration. There are, therefore, stuttering and stammering, and at times a peculiar difficulty of speaking, owing to the attempt being made when the chest is empty; that is, when expiration has just been accomplished. The tongue and lips rarely escape being involved to a considerable extent.

The muscles of mastication and deglutition are generally affected, and hence the food is imperfectly chewed, and often causes choking from difficulty of swallowing it.

In some cases chorea is accompanied with paralysis—the chorea paralytica of authors. This loss of the power of voluntary motion is usually hemiplegic, and involves the same muscles, which are the seat of the irregular movements. Occasionally there are contractions of the limbs, but not to any great degree.

Dr. Weir Mitchell[2] has also called attention to disorderly movements supervening after paralysis, to which he applies the term of post-paralytic chorea. The propositions which he enunciates are :

1. That adults who have had hemiplegia, and who have entirely recovered, are often the subjects of choreal disorder.

2. That the younger the patient the more apt these choreal developments are to ensue.

Dr. Mitchell adduces several interesting cases in support of these propositions, and quite a large number have come under my own observation. But the condition in question is an entirely different affection from athetosis, with which it has been confounded by Charcot, Gowers, and others. The latter, in a very interesting monograph,[1] discusses the question of the identity of athetosis with post-hemiplegic chorea, but fails to discriminate between his own cases, some of which were apparently instances of athetosis, while others were not.

---

[1] *See* the author's "On Certain Causes of Nervous Derangement," for more complete details on this and analogous subjects, and for accounts of other examples.

[2] "Post-Paralytic Chorea," *American Journal of the Medical Sciences*, October, 1874.

Chorea is sometimes of very limited extent. It may be only shown in the hand or foot, but more frequently, when restricted in its topography, it is manifested in the head or face. There may be only a little twitching of the muscles at the angles of the mouth, or of those which raise the upper lip, or of the orbicularis palpebrarum, by which the eyelids are closed, or of the levator palpebræ superioris, or of the corrugator supercilii, or occipito-frontalis. Sometimes the head is rotated suddenly, or twitched to one side, or there is a shrugging of the shoulders.

In several cases that have been under my care, the abnormal manifestations were entirely confined to the organs of voice or speech. In one instance—that of a young girl from Illinois—while there was a general hyperæsthesia of the whole nervous system, there were no choreic movements except of the respiratory and laryngeal muscles. The respiration was therefore exceedingly irregular, and at times inarticulate sounds were made, which were involuntary. Articulate speech was lost from inability to coördinate the muscles, but there was no paralysis, for the tongue could be moved freely in all directions, and the lips were as mobile as ever, except when the patient made an effort to speak. After a few weeks the sound from the larynx was made regularly at each expiration. There were no sounds during sleep.

In this case there was a strong hysterical element present. The affection resisted all treatment, and finally I sent the patient home, scarcely improved except in her general health. One morning she awoke, began to speak, and there was no resumption of the laryngeal sounds. She has continued well ever since, now over two years.

Again, there may be an irregular action of the muscles of speech, and in consequence words are uttered against the will of the patient, and often without any previous knowledge of what is going to be said. Several such cases have been under my observation, and I have alluded to two of them in a recent lecture[2] on chorea. Since then another remarkable case of the kind has come under my care. In this instance there is scarcely a minute during the day that the speech is not going on, and this without the least power on the part of the patient to arrest or direct it. If he is asked a question, he can only use a few apposite words, the others being altogether without relation to the subject about which he wishes to speak.

The convulsive movements in chorea almost invariably stop during sleep. They are also sometimes temporarily arrested by intense mental occupation, but are always rendered worse by emotional disturbance or physical fatigue. On the contrary, they are diminished by mental and emotional quietude.

---

[1] "On 'Athetosis' and Post-Hemiplegic Disorders of Movement," *Medico-Chirurgical Transactions*, vol. lix., 1876.

[2] *Journal of Psychological Medicine*, January, 1871, p. 51.

Strange as it may appear, the sensation of being tired is scarcely ever experienced by choreic patients. Generally, however, there are wandering pains in the limbs, headache, and pain in the back. The cutaneous sensibility is usually increased, but in some cases it is greatly lessened, and may be abolished altogether in some parts of the body.

The functions of the several viscera are ordinarily more or less deranged. There are paroxysms of palpitation of the heart, and the action of this organ is to some extent irregular during the whole course of the disease. Endocardial murmurs are often present, either systolic or diastolic, but are the result of the anæmia which is so prominent a feature of chorea. Respiration is imperfect; the stomach does not digest well; and there are nausea and vomiting. The bowels are constipated; the urine is loaded with phosphates, and is of diminished quantity; and the menstrual function in girls is imperfectly performed, either as regards quantity or quality. The skin is dry and harsh, the hair loses its gloss, the complexion is pale, the lips bloodless, the pupils dilated, and the sclerotic coat of the eye of more than normal whiteness.

The tendency of chorea is to increase to a certain point, and then to gradually diminish. In favorable cases occurring in children, it runs its course in about three months. This period can be materially shortened by appropriate treatment. Sometimes it ceases very suddenly, and in others passes into a chronic condition, which may last for years or during the life of the patient. Occasionally it terminates in death, either directly or in consequence of the supervention of some intercurrent affection. Three fatal cases have come under my observation. One of these I saw several times in consultation with my friend Dr. T. G. Thomas. The patient was a young lady about twenty years of age, and her paroxysms were of the most violent character, sometimes being so strong as to cause her to throw herself off the bed, or to dash about the room with great force. No treatment appeared to exercise any restraining effect, and, after about two years, she died of an abdominal affection. There was no post-mortem examination. In the other two cases, death ensued from exhaustion.

Relapses are common in chorea, especially in children, and sometimes as many as half a dozen attacks occur. Subsequent seizures are usually less severe than the first.

Chorea is often complicated with hysteria—a combination which will be described hereafter. It may also exist in conjunction with rheumatism and malarial fevers, and the exanthemata.

In an interesting monograph, Dr. Gowers[1] gives the results of his studies and investigations relative to certain features of the choreic condition. He found that the electric excitability of both nerves and muscles on the affected side in cases of hemichorea is increased in most cases after the lapse of a few weeks; that there is no necessary relation between the spontaneous spasmodic movements of the affected muscles and the incoördination which takes place when voluntary movements are attempted; that there is no regularity about the distribution of the disorderly movements in cases of hemichorea, and that there appears to be a relation in some cases of chorea with other convulsive affections, such as hysterical and epileptoid seizures of various kinds, and even true epilepsy. As Dr. Gowers observes, this relation, the existence of which is unquestionable, points in some cases to a common origin; in others, to a predisposition excited by the one disease.

Causes.—Chief among the predisposing causes of chorea is age. It is more frequent during the period extending from six to fifteen years than during all the rest of life. Sée, of five hundred and thirty-one cases, found four hundred and fifty-three of ages ranging from six to fifteen years.

During the last ten years, in my hospital and private practice and at my clinics, many cases of chorea have come under my observation and treatment, but I have kept no systematic account of them since the first edition of this work was published (1871). At that time I had full notes of eighty-two cases; of these, sixty-seven were of ages between six and fifteen years. Under the age of six, the disease is less frequent as we go toward birth. Cases have been met with in infants at the breast of six months old. The youngest case I have had was a girl of eighteen months.

After fifteen, the disease, unless it occurs as an epidemic, is not very common. Cases are, however, met with in adults, and even in very old persons. I have seen four cases in individuals over thirty, and three in persons between the ages of twenty and thirty. Of course, I refer to the origination of the disease at these ages: instances of its beginning in childhood, becoming chronic, and lasting through life, are not so rare. In those cases reported by authors of the affection originating very late in life, we have every reason to conclude that they were instances of organic lesions of the brain or spinal cord—probably sclerosis—giving rise to rhythmical movements or paralytic tremor.

The female sex is much more liable to chorea than the male. Of Sée's five hundred and thirty-one cases, three hundred and ninety-three were girls and one hundred and thirty-eight boys.

Of the eighty-two cases of which I have full records, seventy were females and twelve males. Rheumatism has been supposed to be a predisposing cause of chorea. Of one hundred and twenty-eight cases, Sée found sixty-one in association with rheumatism ; but when we come to inquire further, we find that only thirty-two of these were articular rheumatism, while the rest were cases in which there were wandering pains which may have been, and probably were, without the least affinity with true rheumatism.

---

[1] "On some Points in the Clinical History of Chorea." Reprinted from the *British Medical Journal*, London, 1878.

While it is certainly the case that chorea sometimes follows or exists coincidentally with rheumatism, I doubt if its influence is any more than that of a depressing agent to the organism. Of the eighty-two cases observed by myself, only sixteen were connected with rheumatism, while eighteen were just as intimately related to other diseases.

The affection appears to be more common in winter than in summer. Of my cases, fifty-four occurred in the six months from October to March, and twenty-eight in the other six months of the year.

Among the exciting causes, those connected with the emotions occupy the first place. Twenty-seven of my cases were directly the result of fright, apprehension, anxiety, mental excitement, or some other cause of the kind. In eight it was induced by intense study at school, and in four from imitating others similarly affected. This latter factor is not of so general application as in former times, when social life was different. To it is, doubtless, to be ascribed the spread of choreiform movements through certain localities, and especially convents, such as occurred in the thirteenth, fourteenth, and fifteenth centuries, to some of which reference has already been made.

Among other causes, bad hygienic influences and exhausting diseases generally are to be mentioned.

Pregnancy is also asserted to be a cause, and cases are on record in which the fœtus has been born choreic of a choreic mother.

Diagnosis.—There is not much danger at the present day that chorea will be confounded with many of the diseases from which, not long ago, it was not clearly disassociated. Thus, from paralysis agitans, epilepsy, locomotor ataxia, multiple cerebral and cerebro-spinal sclerosis, the fuller acquaintance which we have in recent years acquired of these maladies prevents the necessity of dwelling on their characteristics as distinguished from those of chorea. The course of the latter disease, and the symptoms, other than those connected with motility, are in the others so different that no one who has studied their phenomena could fail in making a correct diagnosis.

With hysteria, some of the forms of chorea may be confounded, and the two affections are not infrequently blended in the same person. It must be confessed, too, that there are cases in which the diagnosis cannot be clearly made out. So far as the patient is concerned, the difficulty of forming a correct opinion in such cases is not a matter of much moment.

The great majority of cases of chorea, such as are met with in children, are readily distinguished from hysteria. The facts of the disease occurring before puberty in so large a proportion of instances, that the emotional system is rarely disturbed as in hysteria, that the affection is not so paroxysmal, and that the accessions of hysteria are more sudden, will be sufficient to render the diagnosis accurate.

From convulsive tremor—with which in some of its forms it is closely analogous—ordinary chorea is diagnosticated by the facts that it is not paroxysmal, but continues while the patient is awake, that the movements are more disorderly, while at the same time more purposive, that the natural tendency is toward spontaneous recovery, and that it usually occurs in children. But it must be admitted that it is difficult to determine to which disease certain rhythmical and paroxysmal disorders are to be ascribed. It would perhaps be more correct to place all such under the head of convulsive tremor or hysteria, with which affections they are certainly closely allied.

Prognosis.—This is usually favorable in those cases which occur before puberty. The chorea of adults is, however, in most instances, a very unmanageable affection, and generally either terminates in death or becomes permanent. Cases in which death has ensued have been reported by various authors—among them, Dr. John W. Ogle,[1] Dr. J. Hughlings Jackson,[2] and Dr. G. Sée.[3] As already stated, three fatal cases have occurred in my own experience. The tendency, however, in the chorea of young persons is decidedly toward recovery, even under unfavorable circumstances as regards hygiene or medical treatment.

Morbid Anatomy and Pathology.—In many cases of persons dying, either from chorea or from intercurrent affection, no changes have been found which could, with probability, be regarded as constituting the disease. In other cases, morbid alterations from the healthy state have been found. The idea has therefore prevailed that there are two kinds of chorea—one which is entirely functional, belonging to the so-called neuroses, the other the result of organic disease of the brain or spinal cord, or both. In Ogle's sixteen fatal cases, congestion of the brain and its membranes was found in some, while in others the disease existed in the spinal cord.

In an analysis of one hundred cases of chorea, Dr. Hughes[4] cites fourteen fatal cases. In all but four of these there was intra-cranial congestion with other structural changes, such as softening, opacities, and adhesions. The spinal cord was not examined in six cases. Of the remaining eight, it was healthy in three, and congested, softened, or with adhesions or opacities of the membranes in the remaining five.

---

[1] "Remarks on Chorea Sancti Viti, including the History, Course, and Termination of Sixteen Fatal Cases," etc., *British and Foreign Medico-Chirurgical Review*, January, 1868, p. 208.

[2] "The Physiology and Pathology of Hemi-Chorea," *Edinburgh Medical Journal*, October, 1868.

[3] *Op. cit.*

[4] "Digest of One Hundred Cases of Chorea," "Guy's Hospital Reports," vol. iv., 1846, p. 360.

In seven fatal cases, collected by Romberg,[1] there was softening and degeneration of different parts of the brain and of the spinal cord.

Other similar cases have been reported, and in the majority there were fibrinous concretions on some portion of the heart's valves or lining membrane.

In 1850 and 1863, Dr. Senhouse Kirkes[2] published the details of a number of cases which went to show the association between chorea and rheumatism, and he made the prediction that "future experience will still more positively demonstrate that an affection of the left valves of the heart, with the presence of granular degeneration upon them, is an almost invariable attendant upon chorea, under whatever circumstances the chorea may be developed." The relation is also insisted upon by Sée and other authors, and such cases as those of Ogle are cited in its support. But the doctrine is only applicable, with any probability, to the fatal cases, and, in those of Ogle, rheumatism was not always an antecedent. In regard to this point, I am entirely in accord with the views expressed by Dr. Ogle in the following extract, which I make from his valuable paper:

"Again it might be asked, if there was merely a mechanical cause (which, of course, would be constant in operation), such as embolism, why should the movements be so decidedly and universally interrupted during quiet sleep? Or, why should certain peculiarities as to age or sex be considered as predisposing influences? Recognizing the frequent existence of these fibrinous deposits, or granulations, on the heart's valves in chorea, I should be much inclined to look upon these post-mortem appearances rather as results of some antecedent condition of the blood, common also to the choreic condition. It is very freely recognized that this affection is frequently in some way or other connected with that condition of blood which obtains in what we call anæmia, or that existing in rheumatic constitutions. In both of these states we know that the fibrine of the blood is much in excess (as also it is in pregnancy and other conditions looked upon as obnoxious to chorea), and in these states we know that the fibrine (with which the blood is surcharged) is very prone to be readily precipitated, either owing to its superabundance or from other obscure and acquired properties (possibly also from some interference with the relation of the fibrine and the other constituents of the blood), upon the heart's walls or valves. May not this hyperinosis be the explanation of the coincidence alluded to? In most cases, the deposit is probably very slight, and, in many cases, so slight as to require search for it. May it not infrequently be that it is often only found in quite the dying state? Speculation might suggest that the fibrinous deposits arise from some interference with the degree of solubility of the fibrine, induced by the presence of some ununited elements within the blood (some result of tissue-metamorphosis) produced by the excessive muscular action and other functional disturbance which exist in the choreic state, thus being not in any way related to this state as a cause, but as a consequence."

---

[1] "Lehrbuch der Nervenkrankheiten," Band ii.

[2] *London Medical Gazette*, 1850, and *Medical Times and Gazette*, 1863.

In the paper to which reference has already been made, Dr. Hughlings Jackson associates hemichorea with the plugging by emboli of the vessels of the corpus striatum of one side, and, in a recent valuable paper, Dr. Charlton Bastian[1] says:

"I need only hint at the important bearing which the possibility of the occurrence of minute embolisms of this kind may have in the elucidation of previously-obscure forms of so-called functional disease of the nervous system, as I hope shortly to publish the details of a fatal case of chorea, in which such embolisms led to ruptures and obliterations of small vessels throughout the corpora striata and in the course of the middle cerebral arteries generally—this being a case of bilateral chorea in which delirium was also present."

As the result of our present knowledge of the morbid anatomy of chorea, while it cannot be said that we are able to define its seat with accuracy, we have strong evidence to support the view that it is not a neurosis or functional affection—if, indeed, there are any such—and that it is the result of changes taking place in the cerebro-spinal system. As previously stated, I am inclined to think that there are at least two distinct diseases—one due to spinal and the other to cerebral lesion, the latter probably consisting of several forms—but that it is advisable to consider them as one disease of various types, until further investigation enables us to speak with certainty on the subject, and to classify them according to the morbid anatomical condition of each.

In the paper already cited, Dr. Hughlings Jackson says of the choreic phenomena: "They are not mere spasms and cramps, but an aimless progression of movements of considerable complexity, much nearer the purposive movements of health. They are not so much incoherences of muscles (like the 'fist' we see in a partial fit of those convulsions, which begin unilaterally where all the muscles of the hand are in action at once) as incoherences of *movements of muscles*. There is some method in their madness. They are not analogous to playing at once many keys of a piano in mere order of continuity, but to a random playing of harmonious chords. Again, they are *successions* of movements; moreover, they are successions of *different* movements."

---

[1] "On the Plugging of Minute Vessels in the Gray Matter of the Brain," etc., *British Medical Journal*, January 30, 1869, p. 96.

Dr. Jackson's theory of chorea is, that it is, like epilepsy, the result of "discharging lesions" of the cortical matter of the cerebrum; and the experiments of Fritsch and Hitzig, Nothnagel, Ferrier, and others, go very far to confirm his views. Two essential points of difference from epilepsy must, however, be noted: the facts that in chorea there is no loss of consciousness, and that the discharges are successive, not paroxysmal, and less automatic. Moreover, his hypothesis leaves out of consideration the spinal element of the disease. That there are discharging and inhibitory centres in the spinal cord, is supported by many artificial and natural experiments. The "spinal epilepsy" of Brown-Séquard is doubtless often a chorea of spinal origin; and my own experiments, cited under the head of convulsive tremor, also show that there are motorial centres in the spinal cord.

Treatment.—Diseases which are almost certain to terminate fatally, and those which ordinarily recover without medical treatment, are very sure to have a great many medicines used in their therapeutics. Chorea belonging, as it does, to this latter category, has a medical armamentarium almost equaling that of hydrophobia. I shall, of course, not even pretend to mention all these measures, but will merely cite those which the weight of evidence, and especially that derived from my own experience, indicates as the most effectual. Of the benefit to be derived from proper medical treatment in shortening the duration of the disease, and preventing chronicity, I have no doubt.

In this country zinc is probably more used in chorea than any other single remedy. I have employed it in many cases, and sometimes with good results. My preference used to be for the sulphate, which I gave in gradually-increasing doses, from two or three grains up to twenty or thirty, three times a day, dissolved in a sufficient quantity of water, to prevent gastric irritation. Latterly, however, I have used the bromide with even better results. Ten drops of a solution of the salt, consisting of a drachm to the ounce, of water or simple syrup, may be given three times a day as an initial dose, the quantity being gradually increased as rapidly as the stomach will bear. When the choreic symptoms begin to disappear, the doses should be diminished in the same gradual manner in which they were increased.

Iron is also frequently administered as a sole remedy, and still more generally as an adjuvant. Indeed, no matter what special treatment may be adopted, iron is generally indicated to improve the quality of the blood. I rarely use it unless for this latter purpose.

Tartarized antimony, copper, sulphate of aniline, Calabar bean, and various other substances, have been employed with more or less success, according to reports, but I have little personal experience of their value, except as regards the Calabar bean, which I have several times employed as an adjuvant, but with doubtful results.

I have used both the primary galvanic and induced currents in many cases. In my opinion they are inefficacious except in that form in which there is distinct paralysis.

Arsenic enjoys a high reputation in the treatment of chorea, and, if properly administered, may be regarded as almost a specific. It should be given in gradually-increasing doses up to the point of inducing evidence of its toxic influence, such as nausea and vomiting and puffiness of the face, especially under the eyes. For a child of five or six years the initial doses may be four drops of Fowler's solution three times a day for the first day; for the next day, three drops are given at a dose; for the next, four, and so on till the phenomena mentioned appear. Then the doses should be set back to four or five drops, and again increased as before. Of the benefits of this treatment no one who has tried it can have any doubt. Its advantages have been shown in a report of cases from the clinique of the University Medical College, made by Dr. Morton.[1] But the gastric method of administering arsenic is not so efficacious in the treatment of chorea as the hypodermic, and in a recent paper[2] I called attention to this point, following Radcliffe, who over ten years ago introduced the practice:

In this country hypodermic injections of arsenic in the treatment of chorea appear to have been first used by Dr. J. Lewis Smith,[3] but since that time the measure does not seem to have attracted any attention.

For the last ten years I have, in obstinate cases of chorea, employed hypodermic injections of Fowler's solution with marked success. In recent or slight cases they do not appear to be necessary, these yielding readily to the use of arsenic by the stomach, or very often getting well of themselves; but in instances of long standing, which are generally classed as incurable, I am quite sure that we have, in the means referred to, a valuable therapeutic measure, which ought not to be disregarded.

In administering arsenic by this method a few points of manipulation are to be considered, for there is a decided tendency to the causation of cellulitis and consequent abscess, and also of painful cutaneous inflammation.

A point for the injection should be chosen in some part of the body where the skin is loosely attached to the subjacent tissues. The skin near the insertion of the deltoid is not a suitable place for the hypodermic injection of arsenic, however well adapted for injections of other substances. I very soon found out that, when inserted there, erythema or abscess, or both, were the invariable sequences. Moreover, the mere act of injecting arsenic in those situations where the skin is tight and the tissues dense is accompanied with very considerable pain.

---

[1] "Treatment of Chorea by Arsenic," *Neurological Contributions*, No. II., p. 79.

[2] "On the Treatment of Chorea with Hypodermic Injections of Arsenic," *St. Louis Clinical Record*, October, 1879.

[3] *Medical Record.*

The best point is on the front of the forearm about midway between the wrist and the bend of the elbow. Here the skin is loose, and can be easily lifted up by the thumb and finger from the tissues below. In the next place the arsenic should be deposited just under the skin in the cellular tissues, and not in the substance of the skin or muscles. Neglect of this point will almost invariably lead to the formation of abscess. The point of the syringe should therefore be just carried through the skin and then for about half, an inch parallel to the face of the arm. The injection should then be made slowly, and it is well to lift up the skin over the place where the injection has been made, so as further to facilitate its absorption.

And, lastly, it will not do to inject the undiluted Fowler's solution, for if this provision be not followed, cellulitis, erythema, and intense pain, will certainly be produced. The dose which it is deemed proper to inject should be diluted with at least an equal quantity of water, or, preferably, of glycerine. The latter substance seems to be more readily absorbed and to produce less irritation than water. All these precautions are for the purpose of preventing local troubles. There is certainly a strong disposition on the part of arsenic to produce them. If, however, attention be paid to the rules I have laid down, there will rarely, if ever, be any local disturbance.

The dose of arsenic administered by hypodermic injection may be very considerably larger than that which the stomach will ordinarily tolerate, and it is just here that the superior advantages of the method are most clearly shown. It is in chronic cases of chorea and certain choreiform affections that large doses of arsenic are especially required, and the effect of such doses in curing the disease is not only generally prompt, but is unassociated with any toxic phenomena. I have frequently given as high as thirty-five drops of Fowler's solution by hypodermic injection as an initial dose. It is very certain that the stomach would not tolerate this quantity. Again, I have often carried the amount taken by the stomach to the utmost bounds of prudence—till the eyes were puffed, and vomiting was almost incessant—and then have continued the arsenic in larger doses by hypodermic injection, with the result of the cessation of all gastric symptoms and the rapid cure of the disorder.

With these introductory remarks I pass to the description of two or three cases in which the beneficial effects of the arsenic administered hypodermically were unquestionable:

Case I.—Mrs. A. C., of Jersey City, consulted me, May 11, 1879, for a spasmodic affection of the muscles of the neck attended with great pain. On examination, I found that the left sterno-cleido-mastoid was the subject of clonic spasm, and that the left trapezius was also similarly involved. As a consequence the head was, every few seconds, jerked round toward the right shoulder, at the same time being drawn backward. It was possible, by a strong effort of the will, to arrest these movements for a half a minute, and at times, when alone and undisturbed, they were less strong and frequent. During sleep they entirely ceased. The affection had come on suddenly some five years previously, apparently as the result of exposure to cold. No therapeutic measures (among which had been electricity, water-cure, and braces of various kinds) had produced the slightest beneficial effect.

In the beginning I administered Fowler's solution in doses of eight drops three times a day, increasing the doses a drop every day. When sixteen drops were reached, the skin around the eyes became puffed, and each dose excited nausea and vomiting. Up to this time there had been a very slight degree of improvement, but I found it was impossible to carry the arsenic far enough when administered by the stomach to get the full effect of the drug. I therefore, on the 20th, administered hypodermically one injection of twenty-five drops diluted with a like quantity of glycerine. On the 21st she received thirty drops, and now there were decided evidences of improvement—the pain was greatly mitigated, and the spasmodic movements were less extensive and less frequent. On the 21st thirty-two drops were given, and on the 22d thirty-five. The amelioration was now still more strongly marked, and by continuing the doses of thirty-five drops till the 25th the pain and the movements were caused to cease entirely. The medicine was now stopped, and the patient has remained to this day free from any spasm. There is still (October 6th) a slight tendency for the head to turn to the right, but this is being gradually overcome, and the power over the formerly affected muscles is complete.

Case II.—Miss H., aged twelve, a young lady from Texas, was brought to me by her mother in the autumn of 1878 to be treated for chorea, with which she had been affected for several months. The muscles chiefly affected were those of the face, both shoulders, and both upper extremities, but at times there was a curious protrusion of the abdomen from the spasmodic action of the erector spinæ muscle.

I at once began the treatment with arsenic and the application of pulverized ether to the spine, the former in doses of five drops of Fowler's solution three times a day, increased a drop every alternate day, and the latter once daily. By the time ten drops of the arsenical solution was reached (which was in ten days), there was decided improvement. The eyes were slightly puffed, but the stomach bore the remedy exceedingly well. I continued the medicine up to fourteen drops without exciting gastric disturbance, and then, as the choreic movements had ceased, I refrained from further increase, but kept on with the doses of fourteen drops for three or four days longer. She then went home cured.

But in May, 1879, she returned to me, with all the choreic symptoms as bad as ever, and her mother informed me they had made their appearance a couple of weeks before without apparent cause. I again tried the arsenical treatment with ether to the spine, which had been so beneficial the year before, but it was now apparent that, from some cause or other, the stomach had become intolerant of the drug, for I found it impossible to administer with safety more than eight drops, and this quantity had no beneficial influence over the disease. I therefore determined to use the hypodermic injections. Twelve drops were the initial dose, the next day thirteen were given, the next fourteen, and the next fifteen. There were no choreic movements after this dose was attained. It was given daily for a week, and then the patient was discharged cured. In all this time there had been no toxic symptoms beyond slight puffing of the face.

Case III.—I. H., a boy eight years of age, was brought to me July 10, 1879, affected with general chorea. The case was a chronic one, having lasted about a year, and had been treated by his physician with a single drop of Fowler's solution administered once every alternate day, and with sulphate of zinc in about as efficacious doses. I began the treatment in this case with hypodermic injections of five drops of Fowler's solution given daily, and every alternate day increased a drop. In ten days thereafter the patient was taking ten drops daily. As by this time great amelioration had ensued, I did not carry the increase further, but, with the view of preventing a relapse, the doses were continued for several days. On the 28th all treatment was suspended, the patient being entirely free from choreiform movements.

In cases of acute chorea, a large number of which I have treated with hypodermic injections of arsenic, smaller doses may be given than when administered by the stomach, and they do not require to be so frequently repeated. Thus it often suffices, for the speedy cure of the disease, to give four drops of Fowler's solution hypodermically every alternate day for a week or ten days, and then to increase the dose to five drops for a like period. I have compared the duration of acute chorea as treated by the gastric and hypodermic administration of arsenic, and have ascertained that the period is shortened one half by the latter method. While admitting that the tendency in such cases is, with hygienic measures, toward a cure, the beneficial effects of the arsenic are none the less evident. I have repeatedly seen the most marked improvement result from a single injection.

In a recent monograph Garin[1] has insisted on the advantages of this method of treating chorea, and has adduced many instances of its good effects.

Quite recently, too, I have made use of the magnet as a therapeutical agent in chorea, and with very immediate and decided results in two cases out of nine in which it was employed. Since the publication of the paper[2] in which these results are recorded, I have used it in eleven other cases without effect, except in one which was cured in the course of a day, while wearing a magnet suspended around the neck, so that the instrument lay against the cervical region of the spine. How far the curative influence in these cases was the result of any special power residing in the magnet, and how far it was due to a strong mental impression produced upon the patient, it is, of course, impossible to say in the present state of our experience. I must confess to some doubt on the subject. It is a measure, however, clearly worthy of continued trials.

As to the employment of strychnia, as detailed in former editions of this work, I am not disposed to recommend it, in view of the excellent results obtainable by the use of arsenic, except in special cases, in which, from some idiosyncrasy, the latter medium is not tolerated. It may also be of service as an adjunct in moderate doses.

The ether-spray to the spine, as employed by Lubilski, Zimberlin, and others, is also an excellent adjuvant. Its effect is immediately quieting, and it may be used two or three times a day for five or six minutes along the whole length of the spine.

In the paroxysmal forms of chorea, ether or chloroform by inhalation is often necessary to cut short or prevent an immediate seizure, but in other respects the treatment mentioned is entirely applicable.

In all cases hygienic measures are of the utmost importance. Exercise in the open air is indispensable ; the food should be of the most nutritious character ; the bedroom should be well ventilated ; bathing should be frequent ; the bowels should be kept well regulated ; and the child, if at school, should be at once removed, and all study for the time be interdicted. Ridicule or threats, so often indulged in toward choreic children, generally do harm, but at the same time they should be encouraged to use all reasonable effort to prevent a bad habit being formed. In the epidemic variety of the disorder, threats, and even strong repressive measures, are, on the contrary, decidedly beneficial in curing and arresting the further progress of the disease.

It is certainly advantageous to keep the patient mentally and physically in a state of comparative repose, but I have never obtained any beneficial effect from confining him to bed in a dark room, as recommended by some writers. On the contrary, I have several times seen the disorder aggravated by this measure. It is one that is particularly distasteful to most children, and hence keeps them in a continual state of fretfulness and excitement. Moreover, it is a measure decidedly antagonistic to the general good health of the patient, who requires light and fresh air as influential hygienic factors in bringing about a favorable result. As regards mental occupation, hard study is of course to be avoided ; but I do not think it advisable to prohibit the reading of such books as amuse, without requiring any considerable degree of intellectual effort for their understanding.

---

[1] "Du traitement de la chorée spécialment par l'arsenic et les injections du liqueur de Fowler," Paris, 1879.

[2] "The Therapeutical Use of the Magnet," *New York Medical Journal*, October, 1880; also *Neurological Contributions*, No. III.

CHAPTER V.

HYSTERIA.

A LARGE volume might be written on hysteria—and many such have been published—and there would still be points in its clinical history unconsidered. It is difficult, therefore, in a general treatise like the present, to give a full view of a disease which plays so important a part in nervous pathology, and which is so varied in its manifestations. The most that I can hope to do is to lay down certain broad principles and features, and leave the recognition of details to the intelligence and discrimination of those who read this work.

Symptoms.—The phenomena of hysteria may be manifested, as regards the mind, sensibility, motility, and visceral action, separately or in any possible combination. Thus it is not uncommon to meet with cases in which the only evidence of the disease is seen in abnormal mental action ; others are characterized solely by derangements of sensibility, such as hyperæsthesia or anæsthesia ; others by aberration of the faculty of motion, such as paralysis, spasms, contractions. Again, all of these categories may be witnessed in the same person, giving rise, among other phenomena, to coma and convulsions ; and again, some one or more of the viscera may be deranged in their functions, and thus the appearance of organic disease be simulated.

As there is such a marked want of uniformity in the character of hysteria as it affects different persons, I will not endeavor to present a typical case of the disorder, but will consider separately the principal phenomena which may have an hysterical origin. But, in setting out to make the attempt, I am reminded of Dante's despair at the thought of his inability to describe the horrors of the ninth gulf :

[1] *Boston Medical and Surgical Journal*, May 30, 1872.

"Chi poria mai pur con parole sciolte
Dicer del sangue, e delle piaghe appieno,
Ch'io ora vidi, per narrar piu volte?
Ogni lingua per certo verria meno,
Per lo nostro sermone, e per la mente,
C'hanno a tanto comprender poco seno."

The Hysterical Diathesis.—Though it is very common to hear the hysterical diathesis or temperament mentioned by medical authors, I have never been able to recognize its existence by any external traits. The fact that it has been so very differently described by writers, from Hippocrates and Galen to our own day, is good evidence that it is not readily detected.

Thus, Hippocrates and Galen recognized the existence of the hysterical temperament, but each gave it different characteristics. Louyer-Villermy[1] had very decided views of its features, and he described it as follows:

"Every hysterical woman is stout, short, dark, plethoric, full of life and of health. The complexion is brunette and ruddy, the eyes black and sparkling, the mouth large, the teeth white, the lips of a carnation red, the hair luxuriant and of the color of jet, the sexual organs well developed, and the spermatic liquid abundant."

Aside from his physiological error relative to the spermatic liquid, these are the characteristics of the women of the south of Europe. If he had lived in the north, where hysteria is fully as common, he would have found that his description of the hysterical temperament would not have held good. Indeed, Sydenham, Whyte, Copland, and other English authors, represent the hysterical predisposition with almost the very opposite characteristics. As Briquet[2] remarks, there is no hysterical constitution appreciable by the study of external appearances. The disease takes women as it finds them, blondes, brunettes, stout, thin, strong, weak, ruddy, or pale, there is no choice. Some hysterical women have delicate figures and intelligent minds, but there are others whose dull, stolid faces give evidence of their stupidity; and others, again, whose thin, fleshless, and wan faces tell us that the Greek type of female beauty is not to be regarded as predisposing to the development of hysteria.

While, therefore, admitting the existence of the hysterical diathesis, I know of no marks by which its presence can be determined, other than the acts of the patient, which go to make up the clinical history.

Mental Symptoms.—These are very various, but generally consist in emotional disturbance, an inability or indisposition to exert the will, and in the existence of illusions, hallucinations, or delusions. Attacks are often characterized by no other prominent symptoms than those connected with mental action, and they may assume every possible character. At times, the patient is depressed in spirits, and sheds tears profusely; a few minutes afterward, she has forgotten her grief, and laughs immoderately, without adequate cause. Sometimes she laughs and cries at the same time.

---

[1] Quoted by Briquet, "Traité clinique et thérapeutique de l'hystérie," Paris, 1859, p. 91.

[2] *Op. cit.*, p. 92.

Or, there may be a total insusceptibility to any emotion, a listless *insouciance*, which contrasts strongly with her natural disposition. Or, again, an emotion the exact opposite of the proper one is excited. This is quite a common form of manifestation. A mother, for instance, is informed that her daughter has contracted an improper marriage, and is immediately seized with immoderate laughter, and shows every expression of pleasure, when the rest of the family are overwhelmed with grief and shame. Another draws the chief prize in a lottery, and begins at once to cry and wring her hands. A third, hearing that burglars have entered the house and have stolen all her jewelry and silver, sits stolidly in her chair, her hands folded in her lap, and her whole expression indicating the most complete indifference. During either of these conditions, she may be entirely silent, or excessively voluble, or she may exhibit other hysterical phenomena.

As regards the will, the manifestations of disorder are sometimes very remarkable. That the patient is, for the time being, unable to exert it, is evident, but, under the influence of some strong exciting cause, she frequently astonishes those about her by suddenly reacquiring her lost volitional power.

A young lady came under my charge for what was supposed to be a disease of the spinal cord. She had taken to her bed suddenly, soon after striking her back rather gently against the edge of a table, declaring that she could not walk. On examination, I was convinced that there was no disease whatever of the spine, other than that of a purely hysterical character, and I so expressed myself to her. She, nevertheless, insisted upon it that her spine was seriously injured, and she continued to keep her bed, lamenting daily her sad fate at being compelled to pass so long a time shut out from the enjoyments of life. There was no paralysis or even simulation of it, for she moved her legs about freely enough in the bed. But, one evening, her brother, who had long been absent, returned home. She heard the bustle in the house attendant upon his arrival, but all were too busy to pay any attention to her in her chamber up-stairs. Suddenly exclaiming, "I can stand this no longer," she sprang from her bed, rang for her maid, and, hurrying on her clothes, proceeded down-stairs and entered the drawing-room, to the great surprise of all the family.

In another case, a lady closed her eyes, and declared that she could not open them. She was brought to me as a case of double ptosis. There was no spasm of the orbicularis palpebrarum on either side, and

I had no difficulty in opening the eyes by gently raising the lids. The pupils were normal; there was no diplopia, and there were no evidences of such cerebral lesions as are generally met with as causes of ptosis. Moreover, she was subject to paroxysms of hysterical syncope. Under the circumstances, I had no hesitation in expressing my opinion to her friends that the case was one of hysteria. I advised the use of the induced current to the eyes, and she found this so disagreeable, not to say painful, that two applications were sufficient to restore her volitional power, so that she opened her eyes without difficulty.

In my remarks on aphasia, I have cited a case (p. 166) in which the power to speak suddenly returned under the influence of excitement, and was suddenly lost again, to be gradually recovered.

Many cases of this loss of volition in hysteria have been under my care, and most physicians have witnessed similar instances.

Illusions are very common phenomena of hysteria, and these may be connected with any or all of the senses. A ball rolling over the floor is taken for a rat; the sound of rain falling on the roof is mistaken for the noise of burglars in the next room; the knives used at table all "smell fishy;" every thing tastes sour or bitter or sweet, as the case may be, and a draught of cold air on the hand is supposed to be the touch of a person or a spirit.

Hallucinations of various kinds are equally frequent. Images are seen where there is nothing; voices are heard where there is absolute silence; odors are smelt where there is nothing to smell; and strange tastes are perceived when the mouth is empty.

Thus one patient sees angels, another demons, another animals of various kinds. One hears voices whispering to her, another musical sounds, and another noises like the breaking of glass or dishes. Another is constantly sensible of a smell as if something is burning, and another always has a taste of turpentine in her mouth.

It is not often the case that these erroneous perceptions impose on the intellect, but sometimes they do, and then delusions are entertained, or these may, as in cases of absolute insanity, be formed without the intervention of the deranged perceptive faculties. They differ however, from the delusions of insanity, such as have been already described, in the facts that they do not last long and that they rarely exercise any powerful influence over the actions of the patients.

Besides these mental phenomena indicative of cerebral disturbance, there are, sometimes, an extraordinary acuteness of understanding and readiness at reasoning and speech quite beyond the natural powers of the patient. At other times, on the contrary, the intellect is dulled, and the conversational power reduced to a low point.

Sensibility.—This may be affected so as to result in the production either of *hyperæsthesia* or *anæsthesia*.

Hyperæsthesia, caused by hysteria, is characterized by the facts that it is never permanently fixed in one place, that it is generally excessively acute, and that it is unaccompanied by evidences of serious disease of the nervous centres or the nerves. A common seat is the skin, and its favorite region is the trunk, especially the skin over the mammary glands, and that covering the labia majora. Another situation frequently affected is the skin of the face.

Cutaneous hyperæsthesia may consist either of spontaneous pain or of tenderness to impressions made upon the surface of the body. Muscular hyperæsthesia, or myalgia, is likewise common. Dr. Inman[1] has investigated this branch of the subject very carefully, and has ascertained that the painful spots correspond to the origins and insertions of the muscles.

Muscular pains due to hysteria are often mistaken for pains of the viscera. Thus the headache which is so frequent a phenomenon of the hysterical condition is very seldom located within the cranium. It may be of very limited extent, constituting the form known as the *clavus hystericus*, or may be of more extensive limits. Its ordinary situations are the frontal regions, occupying, in this case, the occipito-frontalis and corrugator supercilii muscles; the temporal regions, being then located in the temporal muscles; the vertex, being then seated in the tendon of the occipito-frontalis muscle; and the occipital region, in the occipito-frontalis, trapezius, splenius, and complexus. Briquet states that, of three hundred and fifty-six hysterical patients whom he questioned on the subject, three hundred were constantly subject to headache. I have very rarely met with a case of hysteria in which it was not almost constantly present, and never one in which it was not a symptom at some time or other.

Pains are often felt in the muscles of the chest, abdomen, and back. This latter is a favorite situation, especially in the region between the shoulders, and in the muscles on each side of the vertebral column in the lumbar region.

Pains in the joints are common manifestations of hysteria, and they are often mistaken for serious organic disease. When, as is sometimes the case, they are accompanied with contractions of the muscles, the liability to error on the part of the practitioner is increased. Sir Benjamin Brodie,[2] several years ago, pointed out the true nature of certain affections of the joints occurring in hysterical women; and, since his time, others, among whom Barlow[3] and Skey[4] are to be mentioned, have called special attention to the subject. The pain may be attended with swelling, but there is no accumulation of fluid in the cavity of the synovial membrane. The knee is more frequently affected than any other joint.

---

[1] "On Myalgia: its Nature, Causes, and Treatment, etc.," London, 1860.

[2] "Illustrations of Certain Local Nervous Affections," London, 1837.

[3] "A Treatise on Diseases of the Joints," London.

[4] "Hysteria, etc. Six Lectures delivered to the Students of St. Bartholomew's Hospital, 1866," London, 1867.

Quite recently a young lady has been under my charge whose knee had been for two years kept in a steel apparatus for the purpose of preventing motion. Careful examination convinced me that this was a case of hysterical joint. I therefore flexed and extended the limb several times to its utmost limits, told her to throw away the steel rods, and to walk on the leg as much as she pleased. Within six months she walked as well as she ever had, and was even able to waltz with ease, with no other treatment than daily passive movements of the joint.

In regard to these neuroses Meyer[1] has lately communicated much interesting information, and has indicated the leading phenomena which suffice to distinguish them from organic diseases. Thus the pain ceases at night, light handling is more painful than severe pressure, transient swellings are apt to occur, the temperature of the part is subject to changes, there is no tendency to atrophy of the muscles in the vicinity, and they are often cured spontaneously, or by prayer, or by sudden movements of the joint, or by some powerful physical cause.

Neuralgia often has a hysterical origin, and may be in the form of toothache, pleurodynia, sciatica, or pain in the course of any other nerve. The viscera are likewise frequently hyperæsthetic; the stomach, bowels, the kidneys, bladder, uterus, and ovaries, are the organs most frequently affected. And of these the most common seat of hyperæsthesia in hysterical women is the ovary, and, according to Chairou,[2] the left ovary more frequently than the right. I have several times succeeded in causing hysterical attacks by moderate pressure on the ovary, and have rarely failed to find one or both the seat of marked tenderness in cases of the affection. Indeed, so common is it to find ovarian tenderness in hysterical women, that I am almost disposed with Chairou to regard this condition as a pathognomonic sign. Charcot[3] also lays great stress on the symptom.

The organs of the special senses rarely escape having their sensibility exalted, and, consequently, there are increased power of vision, morbid acuteness of hearing, and an abnormal sensitiveness of the smell and taste. Sometimes with these hyperæsthetic conditions there is pain.

Anæsthesia.—Though not so common as hyperæsthesia, anæsthesia is frequently a manifestation of hysteria. One of its most common seats is the skin. In the days of witchcraft, many a hysterical woman with anæsthetic spots on her skin, went to the gallows or the stake on suspicion of being leagued with the devil. The belief was that, wherever the hand of the arch-fiend or his assistants touched the skin, the spot at once lost its sensibility.

---

[1] *Berliner klinische Wochenschrift*, No. 26, 1874. Also *Psychological and Medico Legal Journal*, September, 1874.

[2] "Études cliniques sur la hystérie," Paris, 1870, p. 7.

[3] *Op. cit.*, p. 283.

Two patients are now under my charge in whom there is hemi-anæsthesia, paroxysmal in its character. When it is at its height, no irritation applied to the skin is felt, not even the wire brush of a powerful induction-coil. In neither case are the attacks preceded or accompanied by numbness.

Sometimes the location is very limited, and the loss of sensibility may be partial or complete. In the former case there is numbness, and the full extent can only be exactly ascertained by the æsthesiometer.

The mucous membranes may become anæsthetic. One frequently affected is that which lines the genital canal. In such a case, the sexual passion is entirely extinguished, coition is unattended with pleasure, and may even excite disgust.

The organs of the special senses may be the seat of anæsthesia, and thus blindness, deafness, loss of the senses of smell and of taste, may be caused, more or less complete in character, in different cases.

Chairou[1] has, however, shown that in all cases of hysteria the reflex excitability of the larynx is abolished. If in a hysterical woman the finger be passed down the throat so as to be brought in contact with the epiglottis, it will be found that this part is absolutely insensible, and that it can be rubbed or even scraped with the nail without causing irritation of any kind. Or the superior orifice of the larynx may be similarly treated with the finger or with a probang, a feather, a roll of paper, or any similar instrument, without exciting either cough or efforts to vomit.

Since becoming acquainted with Chairou's observations I have invariably made such an operation as that described a part of my examination of hysterical persons male or female, and have never failed to verify his statements. It is somewhat astonishing that his observations have attracted so little attention.

Anæsthesia of the muscles is occasionally met with, and has, at times, been the occasion of much discussion in medical and theological circles. Many of the phenomena observed in the Jansenist *convulsionnaires* were the result of muscular anæsthesia. In an essay[2] recently published, I have called attention to the symptoms, and have adduced several cases from the records of my own experience. The extent of the anæsthesia is sometimes remarkable. In some of the cases that have been under my care, the most powerful induced currents which it was safe to use, failed to cause pain in the muscles to which they were applied.

---

[1] *Op. cit.*, p. 12.

[2] "On Certain Conditions of Nervous Derangement," New York, 1881.

Alterations of Motility.—These may be evidenced in the way of paralysis or of clonic or tonic spasm.

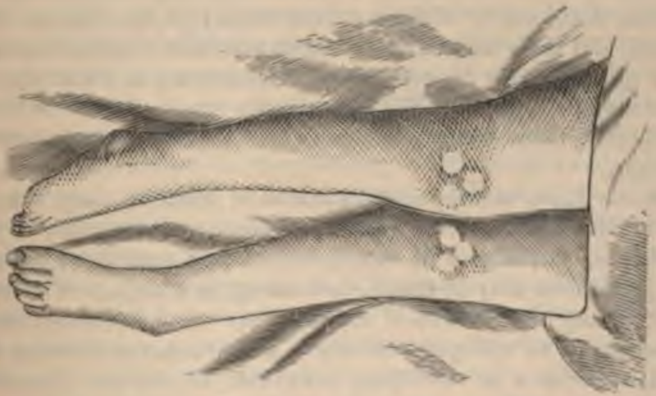
Hysterical paralysis has long been known, and is quite a common manifestation of the affection. It may appear in the character of hemiplegia, paraplegia, or of much more restricted extent. I have a case, now under care, in which it is limited to the index-finger, and I have had several in which a single muscle of the eyeball, or in which the levator palpebræ superioris, was alone affected.

Hysterical aphonia is due to paralysis of one or more muscles of the larynx. Like the loss of power in other muscles from a similar cause, it often comes on very suddenly, and as suddenly disappears.

Paraplegia, hysterical in its character, may be partial or complete as regards a muscle, group of muscles, or a limb. When incomplete, the patient, if it involves the lower extremities, drags her limbs sluggishly along, or shuffles her foot over the floor, using a cane or crutches or holding on to articles of furniture that may be in the room. There is nothing about the gait like that of locomotor ataxia or, in fact, of any other of the diseases of the cord already considered ; and careful observation will generally reveal the fact that, during one interview and examination, the patient walks very unequally, according to the state of her mind at the time, or the influences which act upon her.

Spasms may be either tonic or clonic, and may affect any muscle of the body. In the pharynx, tonic spasm causes the sensation to which the term *globus hystericus* is applied, and which gives rise to the sensation of a ball in the throat. In the œsophagus, spasm may continue for a long time, and may thus simulate stricture. It may also be seated in the stomach, intestines, or bladder.

FIG. 98.



In the limbs spasm of the tonic character causes contraction, and thus, especially when combined with paralysis, may give the appearance of organic lesion. I have frequently known hysterical contractions to last several months at a time, and have had many cases of the kind under my charge in which the actual cautery had been applied to the back for supposed inflammation of the cord.

In some cases the duration is even longer than this. Charcot cites an instance in which a woman, aged fifty-five, was seized, eighteen years previously, with a hysterical paroxysm followed by paraplegia and contraction. At first this latter phenomenon disappeared from time to time to reappear again and again, but for the past sixteen years there had been no change. The extensors and adductors, as will be seen from the accompanying woodcut (Fig. 98), are the muscles mainly affected. The muscles of the legs and thighs were notably atrophied, and the faradaic contractility was lessened. For several years this patient had ceased to exhibit hysterical phenomena.

The subject of permanent hysterical contraction is well considered by MM. Bourneville and Voulet,[1] and the foregoing case is detailed at length in their memoir. In such instances there is probably, as in Case XIII. of their work, in which there was a post-mortem examination, and which has already been cited in this treatise—page 588—symmetrical lateral spinal sclerosis.

Clonic spasms simulate chorea or epilepsy. They are especially common among the women who attend spiritualistic gatherings, and indeed I have seen several cases at such places among the weak-minded men who believe in the nonsense called spiritualism.

The functional actions of the viscera are exceedingly liable to derangement in hysteria. Any organ of the body may be affected, but the stomach appears to be the favorite one. There may be obstinate vomiting, or persistent flatulence, or acidity, or indigestion in some other form; or the bowels may be the seat, giving rise to intestinal indigestion, diarrhœa, or obstinate costiveness; or the kidneys may be involved, and there may be an enormous secretion of pale, limpid urine, or the quantity may be reduced to a minimum; or the uterus or the ovaries may be the seat. Not infrequently organic disease of the heart is simulated, there being palpitation and general irregular action of this organ.

Besides these several manifestations of hysteria, there are paroxysms of the disease, characterized by emotional disturbance, spasm, convulsions, partial loss of consciousness, and sometimes coma. All these phenomena may be manifested during an attack, or a seizure may consist of any one or more of them. The convulsions sometimes bear a resemblance to epilepsy, sometimes to tetanus, sometimes to hydrophobia, sometimes to catalepsy, sometimes to chorea. But, though simulating these diseases, the essentially hysterical paroxysm can be readily distinguished from either of them, mainly by the facts of its lack of consistency, the absence of the constitutional disturbance which attends the others, and by the presence of emotional excitement, and the consequent irrational laughing or crying. Attention will be again directed to some of these conditions in the ensuing chapter.

---

[1] "De la contraction hystérique permanente," Paris, 1872.

Mania may be simulated, but the false can scarcely be mistaken for the real disease by any practitioner with his wits about him.

Causes.—Of the predisposing causes, sex stands first. Of the many cases of hysteria which have been under my charge or seen by me in consultation, but four were in males. In one of these the affection was apparently induced by excessive study, and was characterized by frequent paroxysms of laughing and crying. One was a physician, and the disease took the form of coma; one was a lawyer in this city, the disease in him simulating epilepsy; and the fourth was a shopkeeper from New Jersey, who had tetanoid paroxysms attended with fits of sobbing, crying, and laughing, and in whom it was excited by masturbation.

But, while there is this great predominance of females as the subjects of hysteria, I do not believe that the fact is due to any particular influence of the uterus or other generative organs. It is probably the result of the delicacy of organization, and the greater development of the emotional system, acted upon by the exciting causes to be presently mentioned.

Age is another predisposing cause. The period of life at which hysteria is most common is that extending from sixteen to twenty-five. After the latter age there is a gradual decline until the age is reached at which the menstrual function begins to become irregular, and then the number of cases increases.

The civil condition, as regards marriage or celibacy, is to be taken into consideration among the predisposing causes. Undoubtedly the disease is much more frequent among the single than the married, but it is by no means confined to them. In my opinion the increased proclivity of single women to hysteria is not to be attributed to ungratified sexual desires, or even to the non-fulfillment of the functions of the generative organs, but rather to that lack of aims in life, and the consequent reflection of the thoughts and emotions upon self, which are so inseparably connected with the present condition of single women. Certainly those celibates who have made for themselves objects in existence are no more subject to hysteria, in my experience, than married women. Want of occupation is one of the powerful predisposing causes of hysteria, and it is to a great extent through the direct influence of this factor acting upon a more impressionable organization that, in my opinion, hysteria is more common in women than in men. In those savage and semi-savage countries where women work, hysteria is unheard of. It used to be almost unknown among the negro women in the South, but since their emancipation, if my inquiries have ascertained the truth, it is becoming quite common among them.

Hereditary influence is undoubtedly an important predisposing cause of hysteria. My own statistics are not complete on this point, but they are full enough to show that the majority had either hysterical mothers, aunts, or grandmothers, and many of the others had relatives affected with other nervous diseases. Briquet speaks very emphatically of the decided influence of hereditary tendency as deduced from his inquiries.

The luxurious habits of life attendant upon refinement and education conduce to the development of hysteria. Attendance at theatres and operas, the cultivation of music, the reading of poetry and novels, the study of art, and any other influence capable of developing the emotional system at the expense of the purely physical or intellectual, favor the growth of hysterical tendencies.

Of exciting causes, sudden emotional disturbance ranks first. Anxiety, grief, disappointment, the intense desire of self-gratification, a fit of ill-temper, with other similar factors, often induce paroxysms of the disease. Mental or physical fatigue, menstrual derangement, or uterine or ovarian disorders, may also act as exciting causes.

But probably, above all these, is the contagion set in action by the contact with a hysterical person. I have seen a whole hospital ward of women thrown into paroxysms of hysteria by one patient suffering from an attack.

Diagnosis.—To detail the diagnostic marks which distinguish hysteria from other diseases would require more space than is proper in a work like the present, and would, moreover, be rather a work of supererogation. The physician has simply to recollect that all hysterical affections have a family resemblance, and that, although almost every known disease may be simulated, yet that the counterfeit is never a good one. Attention to the symptoms of the several diseases already, and to be described, with a careful observation of the case, and due inquiry into the antecedents of the patient, will prevent a mistake being made.

He must also recollect that the hysterical patient always tries to impress others with the belief that she is very ill. She craves sympathy, and feeds on it with the effect of nourishing her disease. If she can cajole her medical attendant by appealing to his kindly emotions, she will do it, but failing in this she will try her power over his fears, and will leave no stone unturned to deceive him. Careful watching, with thorough skepticism, will either result in her detection, or in her defeat from sheer weariness.

Prognosis.—As regards the prospect of recovery from any particular manifestation of hysteria, or from a paroxysm of any kind, the prognosis is favorable, provided proper treatment be employed, but, as regards the liability to further attacks, much depends on the circumstances which surround the patient and the time during which she has been subject to the affection. If she can be submitted to proper treatment, without the interference of herself or her friends, the prospect of recovery, even in bad cases, is good; but if she is to be allowed to do as she pleases, or if injudicious friends are constantly lavishing the sympathy and mistaken kindness which keep her disease alive, there is not much use in medicine or hygiene, and, as Reynolds says, the "case is hopeless, and might as well be left alone."

Morbid Anatomy and Pathology.—Hysteria contributes absolutely nothing to the science of morbid anatomy. The brain, the spinal cord, the sympathetic nerve, give no evidence of its former presence. It is true, hysteria very rarely causes death, but hysterical patients have died of intercurrent affections, and post-mortem examinations have been made, and nothing which could reasonably be regarded as the essential cause of the disease has been found. Several of the older writers imagined that they had discovered the lesion in the genital organs, in the stomach and intestines, in the brain, and even in the spleen; but modern research teaches us differently. At present, then, we are in total ignorance of the character of the lesion. From the symptoms, which are so obviously indicative of disordered brain and spinal cord, I have felt myself justified in classing it, provisionally at least, among the cerebro-spinal diseases.

The pathology or morbid physiology of hysteria is beginning to be better understood as our knowledge of the cerebral and spinal actions becomes more complete. Looking at the brain as a complex organ evolving a complex force—the mind—we can understand the possibility of certain parts of it becoming disordered, as regards excess, diminution, or quality, in the results of their actions. We have seen, under the head of insanity, that the mind is made up of certain sub-forces—the perception, the intellect, the emotions, and the will—and that these, when disordered, constitute varieties of insanity, which are easily recognized.

Hysteria essentially consists in the predominance of the emotions over the intellect, and especially over the will, and this exaltation may be so intense as to interfere with the sensibility of various parts of the body, or to derange the contractility of muscles.

At the same time, in the paroxysms of the disease, the reflex and automatic functions of the spinal cord are involved to a great extent.

We daily witness examples of the influence of emotions on sensibility and motility. Fear renders the sensibility more acute and produces trembling, which is simply clonic spasm; grief causes tonic contractions of the muscles; surprise, terror, or horror, paralyzes them; joy or anger destroys sensibility to pain, and so on.

At the same time that there is this exaltation of emotional power in hysteria, the power of the will is not only relatively but is absolutely diminished. The two factors, acting together steadily and persistently, induce many of the manifestations of hysteria. The disease is, therefore, a partial insanity—an insanity, however, in which the patient does not entirely lose the power of control, and which is capable of being overcome by the voluntary effort of the patient, provided a sufficient stimulus to normal volition be brought to bear. It thus happens that, through the influence of such stimulus, every symptom of hysteria disappears as if by magic.

The spinal cord is often secondarily affected, and it is likewise frequently primarily involved. The gray or the white substance, the posterior or the antero-lateral columns may be implicated, the symptoms varying accordingly. Through the spinal cord, in its abnormal condition, we have the convulsions of various kinds, the spasms, contractions, and the paraplegic and hemiplegic phenomena connected with motion and sensation.

As to the influence of the vaso-motor system, though I admit its existence, I am convinced that it is simply a link in the chain, and is secondary to the emotional disturbance already mentioned.

Treatment.—No cases are so well calculated to test the patience and tact of the physician as those of hysteria. For he has an affection to deal with, which not only requires proper medical treatment, but in which he must often exert the highest mental qualities, in order to cure the disease. A great deal, therefore depends on the knowledge of human nature and the force of character of the physician ; and it is doubtless owing to this fact that some physicians, with all their medical knowledge, fail in curing hysterical affections, while others, with no superior science, succeed at once.

The first thing to be done is to gain the confidence and, what is of still greater importance, the respect of the patient. Having done this, any treatment, moral or medical, calculated to relieve her, will be much more apt to produce the desired effect.

During the period between the paroxysms, the treatment must be directed mainly against symptoms. If the patient can be made to believe that her case is thoroughly understood, and that she is not suspected of shamming, and that, with her assistance, the hyperæsthesia, or anæsthesia, or paralysis, will be removed, the effect which is desired will probably be produced. For putting a hysterical patient into a proper frame of mind, I know of nothing equal to the bromides, of either potassium, sodium, calcium, or zinc, given in large doses, repeated three or four times a day, till the full effect is obtained. This, of itself, will generally relieve hyperæsthesia wherever it may be seated, and the influence over the mental phenomena of the disease is usually very decidedly shown.

If anæsthesia be the prominent condition, electricity is to be used, and it is almost a specific. I have never seen a case of hysterical anæsthesia resist it. A few days ago, I was consulted by a young lady who was entirely anæsthetic over the whole of the surface of one side of the body, and who had suffered for several weeks. Three applications of the induced current through the wire brush, which was passed, at each *séance*, over the whole anæsthetic region, entirely cured her.

For hysterical paralysis, strychnia and phosphorus are the best internal remedies. They may be taken together in the form recommended on page 54, and rarely fail to produce a cure. Their effect is, however, greatly increased by the use of electricity, both of the primary and induced forms—the first being applied to the spine, and the latter to the paralyzed muscles.

In cases of spasm, I prefer the bromides, internally, and the primary galvanic current, applied to the contracted muscles.

Visceral derangements are best treated by strychnia and phosphorus, as recommended for paralysis. Counter-irritation, in the form of blisters, is almost always of service. For gastric troubles, the subcarbonate of bismuth, in doses of fifteen or twenty grains, after each meal, will generally prove of service. In a very obstinate case of hysterical vomiting, under my charge, every thing failed but hydrocyanic acid.

Recently, in several extreme cases of hysterical vomiting, and notably in one I saw in consultation with Dr. C. T. Whybrew, I have obtained very prompt results from the valerianate of caffeine in doses of three grains repeated in a half-hour if necessary. Paret[1] adduces several examples of its beneficial effects in like cases.

Hysterical paroxysms are best treated with ether or chloroform, administered by inhalation. I have repeatedly used the hydrate of chloral, but it has not in my hands been as speedy or as effectual in its action as either of the other agents. I give them to the extent of producing complete insensibility, and repeat them again and again, if there are any evidences of a return of the seizure. Whether in the purely emotional paroxysms or those characterized by muscular spasms of various kinds, or any possible combination, nothing is equal, according to my experience, to ether or chloroform by inhalation. I have tried every other known means, from cold water, dashed in the face, to moral suasion, and none of them are comparable to ether or chloroform.

I have also found decided benefit from the mono-bromide of camphor in breaking up what may be called the *status hystericus*. In a recent communication[2] I called attention to its good effects in such cases. It may be given in pill or emulsion in doses of from three to five grains every hour or two, as may be required. In those cases in which ether or chloroform is contraindicated the mono-bromide of camphor is particularly valuable.

---

[1] "De l'emploi de valérianate de cafeine," Paris, 1875.

[2] "Note relative to the Mono-Bromide of Camphor," *New York Medical Journal*, vol. xiii., 1871.

But, for the dissipation of the hysterical tendency, long-continued treatment is necessary. Medicines which are ordinarily regarded as antispasmodics, such as valerian, asafœtida, musk, and the like, I have never seen produce any benefit in any form of hysteria, and, for the purpose of causing any radical change in the organism, they are worse than useless. As medicines for this object, I know of nothing superior to phosphorus, in some one of its forms, and strychnia. They should be taken for months in small doses, and should be supported by all hygienic measures calculated to improve the tone of the system. Travel is of inestimable advantage, and, above all, association with persons of both sexes, whose intellects control their emotions, and who are endowed with sound common-sense and that tact and knowledge of human nature which, for the purposes of every-day life, are of more value than many other qualities often ranked above them.

It is very certain that in most cases of hysteria the exhibition of sympathy is exceedingly injudicious and is generally taken advantage of by the patient to impose still further on those around her. Thus a lady to whom I was called had gotten into a morbid condition attended with frequent paroxysms of weeping, because, as she said, she no longer cared for her husband or children, and that she wished they were dead, etc. All the arguments of her friends failed to convince her that she was a good wife and mother, but, on my telling her husband in her presence that I was afraid it would be necessary to send her to a lunatic asylum, her interest was at once awakened, and the next morning she was entirely free from all hysterical phenomena. She subsequently told me that nothing had roused her but the fear of being put in a hospital for the insane.

In another case a lady had terrified her friends and excited the greatest commotion by threatening to put an end to her life by jumping out of the window. When I saw her she was strapped down to a bed and was being supplicated by half a dozen people in the room not to kill herself, to which she was energetically replying that she would. I loosened the straps, opened the window, and told her to jump out. She walked to the window, looked out for a moment, and then, applying no very polite epithet to me, went back to bed, and I heard no more of her suicidal desires.

A still more remarkable case is given by M. Charcot.[1] The patient, a woman, had been for at least four years the subject of contraction of one of the lower extremities, as shown in the woodcut (Fig. 99). In consequence of her insubordination on one occasion, he spoke to her very sharply, and threatened to send her out of the hospital. The next morning the contraction had entirely disappeared. In the face of facts like these it appears absurd to invoke supernatural agencies.

---

[1] "Leçons sur les maladies du système nerveux," Paris, 1872–'73, p. 313.

It is, perhaps, scarcely necessary to state that the society of other hysterical persons must be rigidly eschewed, and that even the casual meeting with such individuals is dangerous.

FIG. 99.

CHAPTER VI.

HYSTEROID AFFECTIONS—CATALEPSY, ECSTASY, HYSTERO-EPILEPSY.

THERE are certain disorders so very like hysteria in some of its manifestations, and often existing with it in the same individual, that they might with propriety have been considered in the last chapter, especially as by some high authorities the scope of hysteria is so enlarged as to be made to embrace them within its limits. But, though they may owe their existence to the same peculiar condition of the nervous system, to which the ordinary phenomena of hysteria are due, there is sufficient individuality about them to warrant their being studied separately. At the same time there will be no difficulty in our bearing in mind that they are decidedly of such general and special characteristics as to impress us very forcibly with the idea that they are essentially hysterical. We may, therefore, with propriety, class them together in the present chapter as hysteroid.

I.

CATALEPSY.

Although there are no post-mortem appearances characteristic of catalepsy, the phenomena of the disease observed during life point to its seat in the brain and spinal cord. Like epilepsy, therefore, it is a symptom representing an unknown morbid change in the nervous centres.

Symptoms.—Catalepsy is an affection marked by the occurrence of peculiar paroxysms at regular or irregular periods. The seizures usually come on with suddenness, and are characterized by more or less complete suspension of mental action and of sensibility, and by the supervention of muscular rigidity, causing the limbs to retain, for a long time, any position in which they may be placed. The phenomena, therefore, relate to the mind, to sensation, and to motion.

The suspension of mental action is, in general, complete, but in some cases there are an imperfect consciousness and an ability to appreciate strong sensorial impressions. Thus, in a case quoted by Dr. Chambers from Dr. Jebb—which, however, was clearly a case of catalepsy complicated with hysteria—the patient, before emerging from the paroxysm, sang "three plaintive songs in a tone of voice so elegantly expressive, and with such affecting modulation, as evidently pointed out how much the most powerful passion of the mind was concerned in the production of her disorder, as indeed her history confirmed."[1]

The aspect of a cataleptic patient is very striking. The eyelids are sometimes wide open, at others gently closed; the pupils are dilated, and do not respond to strong light; the respiration is slow, regular, but generally so feeble as to be perceived with difficulty; the pulse is usually almost imperceptible, but is rhythmical and sluggish; the face is pale, the mouth is half open, and the rigidity of the body and the coldness of the extremities add to the death-like appearance which impresses all beholders.

The cutaneous sensibility is ordinarily completely abolished. Pins may be stuck into the skin, and they are not felt; but, owing to the abolition of the power of motion and of reflex action, it is possible that in some cases, at least, the patients would give evidence of sensation if they could. Cases are on record in which tears have been caused by excessive emotional disturbance excited by the words or actions of persons surrounding the patients, thus showing that the senses of sight and hearing were capable of being exercised. Such instances are, however, rare, and are probably imperfectly-developed paroxysms, or those complicated with hysteria or ecstasy.

The symptoms relating to the muscles are very remarkable. Coming on, as the paroxysm usually does, without warning of any kind, the patient is at once arrested in any act which is being performed, and the whole body assumes a condition of extreme rigidity. The power of the will over the muscles is lost, and the limbs preserve any position in which they may be placed by the by-standers. Thus, if the arm be raised from the side, it remains extended, and may keep this position for an hour or longer before it sinks slowly back to its original situation. No matter how awkward or irksome the position may be, it is retained till the exalted irritability of the muscles becomes thoroughly exhausted.

---

[1] Article "Catalepsy," in Reynolds's "System of Medicine," vol. ii., p. 100.

The ability to swallow is not lost, and the electric contractility of the muscles is not perceptibly affected one way or the other.

The paroxysm may last a few minutes or hours, or may be prolonged to several days.

The temperature of the body, in all the cases that have come under my observation, was reduced from two to four degrees below the normal standard, and in the extremities much more than this.

The paroxysm generally disappears with as much abruptness as marked its accession. A few deep inspirations are taken, the eyes are opened, or lose their fixedness, the muscles relax, and consciousness is restored. In fully-developed seizures the patient has no knowledge of what has occurred during the attack.

Ten cases of true catalepsy, uncomplicated either with hysteria or ecstasy, have been under my professional care. In two of these the seizures were more or less imperfectly developed, and strong sensorial excitations were, in a measure, perceived and recollected after emergence from the attack. But in every instance the character of the impression was misinterpreted. A bright light thrown upon the eyes with a mirror was spoken of as an "angel's wing which brushed across my face," and the scratch of a pin was remembered as "a piece of ice being drawn over the skin."

In these cases there was the consciousness of mental action during the paroxysm, but it was difficult for the patients to describe the thoughts which took place. They appeared to be somewhat of the nature of dreams. In both cases the muscular rigidity was well marked but was not excessive, and appeared to be mainly manifested in the extensors. It was not difficult to extend the arm or the leg, but flexion required the exertion of a good deal of strength.

In the other eight cases the paroxysms were completely formed. Consciousness was entirely abolished; there was no sensibility anywhere, and no reflex actions could be excited except those of deglutition. In one of these cases, seizures several times occurred in my consulting-room, and I had the opportunity of ascertaining the effect of electricity. If the arm was extended, the strongest induced current I could apply to the biceps, though causing contraction, failed to procure flexion, but relaxation of the extensors was at once produced by the application to them of the primary current.

I likewise, in this case, repeatedly examined the fundus of the eye with the ophthalmoscope, and invariably found the choroids pale, and the retinal vessels straight and attenuated.

In none of these cases was there any knowledge of what passed during the paroxysms, and no consciousness of there having been any mental activity.

Besides these, several instances have occurred in my experience in which cataleptic phenomena were exhibited in the course of other diseases. In one of them, a young man whom I saw in consultation with Dr. Max Herzog, of this city, there was well-marked mania—a second attack. On my entering the room in which he was seated I observed that he had a rapt expression of countenance, and that his limbs were quiet, and apparently rigid. In an undertone I remarked to Dr. Herzog that the patient had a somewhat cataleptic appearance. Seizing his arm I raised it from the body and it remained extended; the other arm was also elevated and continued in that position. I then lifted the legs alternately from the floor, and they were kept in their apparently uncomfortable positions. During the consultation, probably a half-hour, the extremities remained as I had placed them. A few days afterward, he became so violent that it was necessary to send him to a lunatic asylum.

In another case the patient, a young lady of this city, was brought to me by her father for examination and advice. As she entered my consulting-room, I saw that there was a high degree of mental exaltation present—her eyes were raised to the ceiling, her hands were clasped, and her lips were moving as if in prayer. I raised her left arm from the body, and then the right; both remained extended, and continued so till I changed the positions, which I did by bending the elbows, bringing them to the front, putting them behind her, and so on. I then again extended them, and she left the house with them in this position; but, on getting into the street, and feeling a cold wind that was blowing at the time, they fell to her side and she began to use them to draw her shawl around her. She had been subject to epilepsy for several months, but had never before exhibited cataleptic phenomena.

In the former of these cases there was no possibility of ascertaining the mental associations of the patient with the muscular rigidity; in the latter the patient said that she had a very distinct recollection of my extending her arms, but why she had kept them so she did not know, and that she was not conscious of fatigue, or of any other sensation.

It will have been noticed that in both these cases the paroxysms were not spontaneous, but were excited by outside interference.

The particulars of a very interesting case of catalepsy have been recently given to me by Dr. M. B. Early, late house-physician to Bellevue Hospital.

The patient, a German, a cigar-maker, aged twenty-three, had served in the army, entered the hospital October 4, 1872. In the previous July he had been drunk, and, quarreling with some rough people, was severely beaten and kicked on the head and other parts of his body.

On the 27th of September he had an attack resembling a convulsion. He was smoking at the time, and, while thus engaged, his mother noticed that the cigar began to shake, then his whole body quivered. She attempted to take the cigar from his mouth, but the jaws were tightly closed, and the cigar was bitten through. He swallowed the portion that was left in his mouth. He seemed to be conscious, for when requested by his mother to go to bed he shook his head. He did not sleep, but, when spoken to, nodded or shook his head in assent or dissent as the case might be. He did not foam at the mouth or bite his tongue. His feet were very cold.

The attack lasted about five minutes. He then vomited the piece of cigar he had swallowed, and went to bed, sleeping all afternoon.

The following day he had a similar attack, not so severe as the first. During the five following days he was free from paroxysms, but would not talk, although he ate and seemed to understand what was said to him, and would do any little thing his mother requested. On the sixth day, soon after breakfast, he had another paroxysm, but of a different character from the others. While the previous seizures were characterized by tremor, this was marked by a rigidity of all the voluntary muscles in the body. The attack lasted a few minutes, and the next day he was taken to the hospital, where he came under Dr. Early's observation.

On admission, October 4th, he lay in a stupid condition, his eyes sometimes open and sometimes closed. Occasionally he looked around, and appeared to understand what was said to him, but could neither speak nor move. The pupils were dilated. When his limbs were placed in any position they continued there for a considerable period. The muscles were rigid, temperature 100° Fahr.

On being slapped smartly on the buttocks with a book, the patient got up, looked about him, and walked around the ward. He then drank a glass of milk and went back to bed. Just before getting up he smiled, and answered a question. During the night he went to the water-closet. In the morning he arose, looked around him, and drank some more milk. When slapped with a book shortly afterward, he did not move a muscle; seemed more stupid, did not swallow when food was placed in his mouth, and apparently did not feel the prick of a pin.

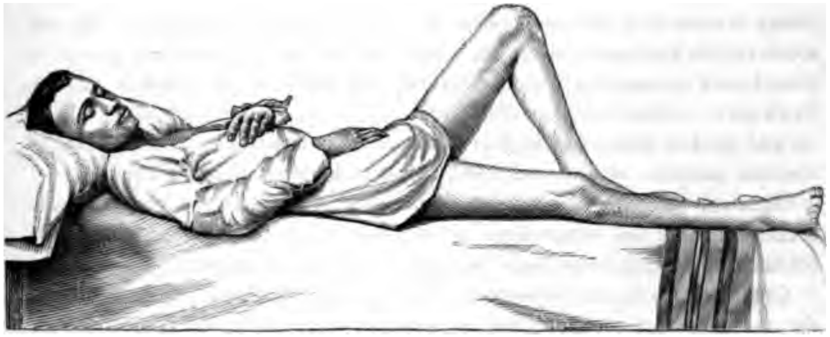
The patient continued in this state for several days. On the 12th he was photographed. The accompanying woodcuts, Figs. 100 and 101, show the positions of his limbs at the time.

Under the treatment the patient gradually improved, and on the 9th of November was discharged cured.

An ophthalmoscopic examination, made November 3d, showed an anæmic condition of the disk.

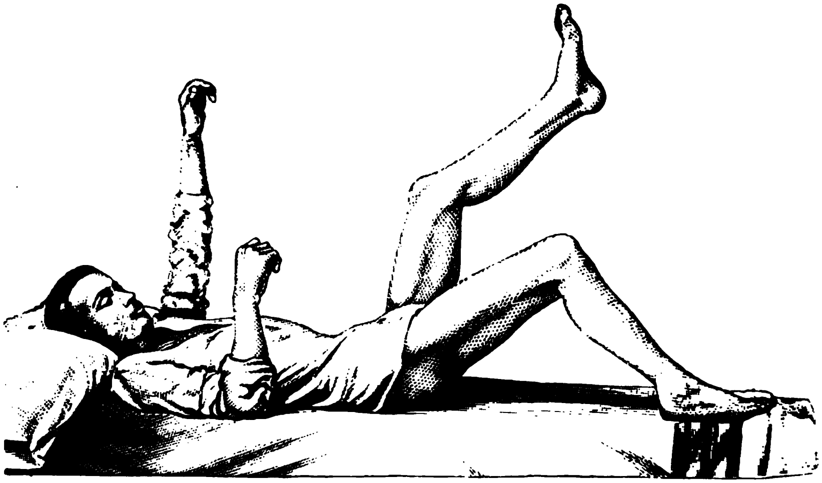
Cataleptic persons are usually of dull and sluggish mental and physical organization. Such has certainly been the case in all the instances that have come under my observation. The disease does not ordinarily show any decided tendency to become worse, either as regards the severity or frequency of the paroxysms, providing the exciting causes be avoided. On the contrary, there is often a well-marked natural tendency to spontaneous cure, or at least to a cure through the influence of purely hygienic influences, moral as well as physical.

FIG. 100.



In the majority of cases catalepsy is complicated with hysteria or ecstasy, and sometimes with epilepsy. Of this latter combination I have seen two cases, and in one of these ecstasy was also a feature. This case I have alluded to in another communication.[1] The patient

FIG. 101.



---

[1] "The Physics and Physiology of Spiritualism," New York, 1871, p. 55.

was a young girl, was cataleptic on an average once a week, and epileptic twice or three times in the intervals. Five years previously she had spent six months in France, but had not acquired more than a very slight knowledge of the language—scarcely, in fact, sufficient to enable her to ask for what she wanted at her meals. Immediately before her cataleptic seizures, she went into a state of ecstasy, during which she recited poetry in French, and delivered harangues about virtue and godliness in the same language. She pronounced at these times exceedingly well, and seemed never at a loss for a word. To all surrounding influences she was apparently dead; but she sat bolt upright in her chair, staring at vacancy, and her organs of speech in constant action. Gradually, she passed into the cataleptic paroxysm, in which she usually remained for from one to three hours. Many cases of the combination of catalepsy with hysteria and ecstasy have become celebrated in other relations than those of true science.

Causes.—Among the predisposing causes, sex is, in my experience, the most efficient, though other writers have denied any influence due to sex. Of one hundred and forty-eight cases cited by Puel,[1] sixty-eight were males and eighty females. Seven of my cases were in females. Hereditary influence is generally apparent. Of the ten uncomplicated cases under my observation, all had relatives affected with some well-marked disease of the nervous system. In four cases, there were near relatives insane; in three, the mothers were hysterical; in one, a brother was epileptic; in one, the father was similarly affected; and, in one, a sister was cataleptic. It rarely begins after the age of twenty-five. Of exciting causes, emotional disturbance stands first. Four of my cases were directly the result—one of fright, one of anger, one of grief, and one of the shock caused by a boy starting out suddenly from behind a door where he had been concealed. In one other case, the cause was worms in the intestinal canal; in two, business troubles; in one a severe fall; and, in the other two, I could not ascertain with certainty what the cause was, though I had strong reasons for suspecting it to be masturbation.

The **Diagnosis** is not a matter of the least difficulty to any one who has even an imperfect knowledge of the phenomena, except, perhaps, as regards its discrimination from hysteria, that simulator of almost every nervous disease. In those cases complicated with hysteria, the distinction is of no importance; in others, the uniformity of the characteristics which indicate catalepsy, with a consideration of the general history of the case, will serve to make the diagnosis sufficiently precise. It must, however, be borne in mind that the two diseases are near of kin, and that the discrimination is important more as a matter of abstract science than as one of any bearing on the therapeutics. It is, however, sometimes a matter of moment to distinguish between the cataleptic paroxysm and death. In former times, instances were not uncommon in which the mistake was made, to be discovered after life had really become extinct in the coffin. Such fatal errors would probably be impossible now with the stethoscope for examining the heart, the thermometer for determining the temperature, electricity for acting on the muscles, and, above all, the ability to place the limbs in positions which they maintain against the laws of gravity. Moreover, our knowledge of diseases in general is such as to enable us to determine with great certainty the course they are liable to take, and the manner in which death occurs in each.

---

[1] "De la catalepsie," "Mémoires de l'Académie de Médecine," tome xx., 1658, p. 409.

Prognosis.—This is usually favorable, even in severe cases. All my patients recovered under the treatment to be presently mentioned.

Morbid Anatomy and Pathology.—There is not much to say relative to the morbid anatomy of catalepsy. In some cases in which death has taken place, other diseases were present, and the lesions found were rather to be associated with them than with catalepsy.

Puel,[1] in his very elaborate treatise, says that the first report of a post-mortem examination of a patient dying while subject to the disease in question is that of Hollerius, made in 1596. The patient, a man, had but one paroxysm, and died the same day. The lungs and liver were gangrenous, a collection of reddish serum was found in the posterior part of the brain, and sanguineous concretions (thrombi) in the superior longitudinal sinus.

Deidier, in 1811, reported the case of an elderly man who had but one paroxysm, lasting a day, and who died eight days afterward. In this instance there were found, on each side of the longitudinal sinus, two little glandular bodies which were described perfectly, and to which the catalepsy was attributed. These were nothing more than the granulations of the dura mater, now known as the Pacchionian bodies.

In a maniac who was subject to catalepsy and who died at Charenton, in 1834, the report by Georget and Calmeil states that the pia mater was found thickened and injected; the cortical substance of the brain was reddened and softened, and the white substance contained enlarged vessels. In another case the same observers found the cortical substance discolored, and the white tissue injected. As they remark, however, these are the lesions of insanity with general paralysis.

In other cases no alterations which could normally be associated with the cataleptic phenomena were discovered.

The pathology of catalepsy is very imperfectly known. The symptoms indicate that the brain and spinal cord are involved, and there is some evidence to show that they are in a state of anæmia. But there is a condition induced in these organs which is the essential feature of the disease, and of this we know nothing. There is a possibility that the affection may be a masked form of epilepsy, and this view is borne out by the fact that the treatment which is most successful in this latter disease is most efficacious in catalepsy.

---

[1] *Op. cit.*, p. 518.

But recent researches have served to give us perhaps some inkling of the real nature of catalepsy, and to supply us with examples of artificially-induced cataleptiform phenomena which are of great interest as analogical to instances of the natural disease. The investigations which have been made relative to motor centres in the brain lead us to suppose that there are likewise inhibitory centres in the cerebro-spinal system, probably both in the brain and spinal cord. We often meet with cases in which there is complete paralysis of one or more parts of the body, and which are suddenly caused by some strong impression produced upon the emotions. Now, catalepsy is, for the time being, a paralysis of the will, a condition in which, while the muscles have not lost their power to contract, there is a loss of volitional influence over them. They are still capable of responding to stimulation from without, but, in the absence of stimulation from within, they retain whatever degree of contraction may be given to them.

Some of the results which follow experiments made to induce what is called the hypnotic state, are very suggestive of catalepsy. A crawfish, as Czermack[1] has shown, can be thrown into the cataleptic condition, during which he is rigid and immovable. And I have repeatedly put frogs, lobsters, and hens, into a similar state. The full consideration of these interesting phenomena would be out of place in a practical treatise on diseases of the nervous system.[2]

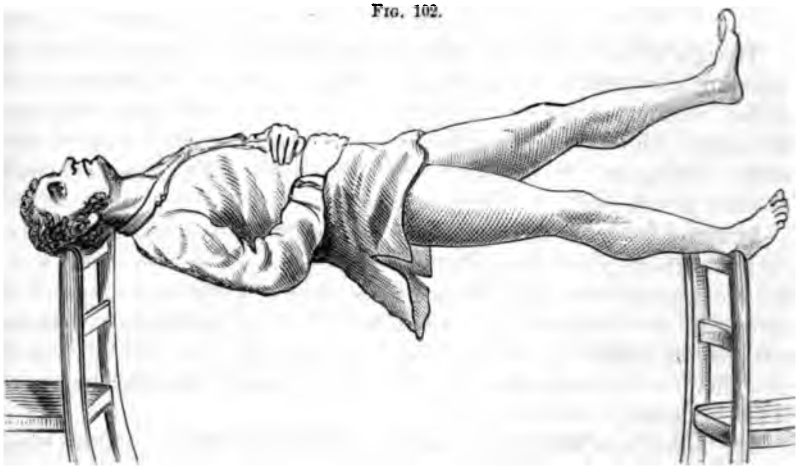
But I may at least state that I have recently developed the most intense cataleptoid phenomena in several subjects through the influence of suggestion, while they were in the hypnotic, or, as I think it should more properly be called, the syggignostic (*συγγιγνώσκω, to agree with*) condition. Among other exhibitions of the phenomena is one which is especially striking. By merely telling the subject that his body is so rigid that he cannot bend it, he at once becomes cataleptic in every voluntary muscle, and may then be laid upon the backs of two chairs, as shown in the cut (Fig. 102), in which position he will remain for several minutes; then the muscles gradually become unable longer to endure the strain, and the body sinks slowly to the floor. Very few trained gymnasts could perform this feat at all, and no one in his normal state could maintain the necessary muscular tension as long as the physically weak young man from whom the drawing is made. As is seen, the body rests only on the occiput and on one os calcis; and I have known the position to be steadily kept for full five minutes. In these cases there is no excitation of muscular contraction by reflex action, such as is supposed by Charcot and Heidenhain to produce it, but it is induced solely by suggesting to the subject that his body is in a rigid state. Immediately the muscles become tense, and he can be handled like a board.

---

[1] "On Hypnotism in Animals," translated from the German by Clara Hammond, *Popular Science Monthly*, September and November, 1873.

[2] For a more complete account of the phenomena and physiology of catalepsy, ecstasy, somnambulism, etc., the reader is referred to the author's work "On Certain Conditions of Nervous Derangement," New York, G. P. Putnam's Sons, 1881.

FIG. 102.



There may thus be in catalepsy inhibitory lesions, just as in epilepsy there are discharging lesions. But as in this latter disease there is something more than the convulsive movements, so in catalepsy there is a morbid element in addition to the muscular inhibition. And this appears to be an overwhelming inclination to agree with the suggestions received from other persons. Catalepsy is therefore hypnotism —or, as I prefer to call it, from its main characteristic, syggignoscism —with the addition of phenomena of muscular rigidity. Any syggignostic subject can be thrown into a cataleptic condition, and the cataleptic patient can readily be made to exhibit the ordinary manifestations of syggignoscism.

Treatment.—The bromide of potassium, or one of the other bromides previously mentioned under the head of epilepsy, is the most efficient agent in the treatment of catalepsy. I have never yet failed to cure the disease with this remedy, combined with the oxide of zinc, and with the simultaneous use of strychnia and other tonics. I have never, however, had occasion to give it in larger doses than twenty grains, three times a day, or to continue it beyond eight months.

In no disease of the nervous system, not even excepting hysteria, is it more necessary that the mind should be brought under proper discipline, and kept as far as possible from the operation of all causes calculated to promote emotional excitement. At the same time, a well-regulated system of hygiene, as regards all the physical requirements of the body, is indispensable.

II.

ECSTASY.

Though closely allied to catalepsy, ecstasy differs from it in several important particulars. One of the main points of difference is, that the patient recollects the train of thought which has been going on during the seizure, and this of itself is sufficient to warrant their being separately considered. It often happens, however, that the two diseases alternate or coexist.

Symptoms.—In ecstasy there is muscular immobility rather than rigidity, although the latter is sometimes present; the eyes are open, the lips parted; the face is turned upward, the hands are often outstretched; the body is erect and raised to its utmost height, or else is extended at full length in the recumbent posture. A peculiar radiant smile illumes the countenance, and the whole aspect and attitude is that of intense mental exaltation.

The mind is so filled with some particular train of thought, that excitations of the senses, if of moderate intensity, are not perceived. We meet with this fact often in normal conditions, when the mind is deeply engaged in reflection, or when it is engrossed with some powerful emotion.

Sometimes there is complete silence, the mind being apparently absorbed with meditation or with the contemplation of some beatific vision. Again, there may be mystical speaking, prophesying, singing, or the lips may be in motion as if in speaking, but without any sound escaping.

At times various attitudes are assumed which are in consonance with the ideas passing through the ecstatic's mind. Again, stigmata or spots of blood appear in the hands or other parts of the body, and which are supposed to represent the wounds made by the nails in the hands and feet of Jesus, or the thrust of the spear in his side; and, again, a real or assumed abstinence from food exists.

Among the ecstatics of a former period, St. Francis of Assisi, St. Catherine of Sienna, St. Theresa, Joan of Arc, and Madame Guyon, are to be mentioned, and whole sects, both among Catholics and Protestants, exhibited all the manifestations of the disorder.

Most of the religious impostors who have at various times made their appearance, and many very sincere and devout persons, have been ecstatics.

In its combinations with catalepsy, chorea, and hysteria, ecstasy has frequently played an important part in the history of the civilized world—at one time, leading to a belief in witchcraft; at another, to demoniac and angelic possession; at another, to mesmerism and clairvoyance; and, in our day, to spiritualism. The consideration of these follies, though interesting, scarcely comes within the scope of the present treatise.

But within the last few years several very remarkable examples of ecstasy have been observed, and some references to two or three of them will probably not be out of place.

First among them, as well on account of the interesting phenomena manifested as from the fact that the patient was regarded by a great many religious enthusiasts—physicians among them—as the subject of miraculous interference, must be placed Louise Lateau.[1] Without going into the full details of the case, a short account will probably prove both interesting and instructive :

Louise Lateau was born at Bois-d'Haine, a small village in Belgium, on the 30th of January, 1850. She was reared in the utmost poverty, was chlorotic, and did not menstruate till she was eighteen years old. She loved solitude and silence, and when not engaged in work—and she does not appear to have labored much—she spent her time in meditation and prayer. She was subject to paroxysms of ecstasy, during which, as many other ecstatics, she spoke very edifying things, of charity, poverty, and the priesthood. She saw St. Ursula, St. Roch, St. Theresa, and the Holy Virgin. Persons who saw her in these states declared that, while lying on the bed, her whole body was raised up more than a foot high, the heels alone being in contact with the bed.

The stigmatization ensued very soon after these seizures. On a Friday she bled from the left side of her chest. On the following Friday this flow was renewed, and in addition blood escaped from the dorsal surface of both feet; and on the third Friday not only did she bleed from the side and feet, but also from the dorsal and palmar surfaces of both hands. Every succeeding Friday the blood flowed from these places, and finally other points of exit were established on the forehead and between the shoulders.

---

[1] For the theological view of this remarkable case the reader is referred to the following works, a part only of those written in support of her pretensions: "Louise Lateau de Bois-d'Haine, sa vie, ses extases, ses stigmates; étude médicale," par le Dr. F. Lefebvre, professeur de pathologie générale et de thérapeutique à la université catholique de Louvain, etc., Louvain, 1873; "Les stigmatisées Louise Lateau de Bois-d'Haine, sœur Bernard de la Croix, etc.," par le Dr. A. Imbert-Gourbeire, professeur à l'école de médecine de Clermont Ferrand, Paris, 1873; "Biographie de Louise Lateau, la stigmatisée de Bois-d'Haine," par H. Van Looy-Tournai, Paris and Leipzig, 1874; "Louise Lateau la stigmatisée de Bois-d'Haine d'après des sources authentiques, médicales et théologiques," par le professeur docteur A. Rohling, translated from the German by Dr. Arsène de Noné, Bruxelles et Paris, 1874; "Louise Lateau, ihr Wunderleben und ihre Bedutung im deutscher Kirchenconflicte," von Paul Majuncke, Berlin, 1875.

Among the treatises in which the miracle is denied, and the phenomena attributed to either disease or fraud, are: "Louise Lateau, Rapport médicale sur la stigmatisée de Bois-d'Haine fait à l'académie royale de médecine de Belgique," par le Docteur Warlomont, Bruxelles and Paris, 1875; "Science et miracle, Louise Lateau, ou la stigmatisée belge," par le Dr. Bourneville, Paris, 1875 "Les miracles," par M. Virchow, *Révue des cours scientifiques*, January 23, 1875

At first these bleedings only took place at night, but after two or three months they occurred in the daytime, and were accompanied by paroxysms of ecstasy, during which she was insensible to all external impressions, and acted the passion of Jesus and the crucifixion.

M. Warlomont, being commissioned by the Royal Academy of Medicine of Belgium to examine Louise Lateau, went to her house, accompanied by several friends, and made a careful examination of her person. At that time, Friday morning at six o'clock, the blood was flowing freely from all the stigmata. In a few moments the sacrament would be brought to her, and then the second act of the drama would begin. The scene that followed can be best described in M. Warlomont's own words:

"It is a quarter-past six. 'Here comes the communion,' said M. Niels [a priest], 'kneel down.' Louise fell on her knees on the floor, closed her eyes and crossed her hands, on which the communion-cloth was extended. A priest, followed by several acolytes, entered; the penitent put out her tongue, received the holy wafer, and then remained immovable in the attitude of prayer.

"We observed her with more care than seemed to have been hitherto given to her at similar periods. Some thought that she was simply in a state of meditation, from which she would emerge in the course of half an hour or so. But it was a mistake. Having taken the communion, the penitent went into a special state. Her immobility was that of a statue, her eyes were closed; on raising the eyelids the pupils were seen to be largely dilated, immovable, and apparently insensible to light. Strong pressure made upon the parts in the vicinity of the stigmata caused no sensation of pain, although a few moments before they were exquisitely tender. Pricking the skin gave no evidence of the slightest sensibility. A limb, on being raised, offered no resistance, and sank slowly back to its former position. Anæsthesia was complete, unless the cornea remained still impressionable. The pulse had fallen from 120 to 100 pulsations. At a given moment I raised one of the eyelids, and M. Verriest quickly touched the cornea. Louise at once seemed to recover herself from a sound sleep, arose and walked to a chair, upon which she seated herself. 'This time,' I said, 'we have wakened her.' 'No,' said M. Niels, looking at his watch, 'it was time for her to awake.'"

She remained conscious; the blood still continued to flow; the anæsthesia had ceased, her pulse rose to 120, and at the end of half an hour she was herself. "Our first visit ended here. At half-past eleven we made another. The poor child had resumed her attitude of extreme suffering, against which she contended with all the energy that remained to her. The wounds in the hands still continued to bleed. M. Verriest auscultated with care the lungs, heart, and great vessels, and found the *bruit de souffle* which he had detected in the morning at the apex of the heart and over the carotids. The handle of a spoon pressed against the velum, the base of the tongue, and the pharynx, provoked no effort at vomiting. The glasses of our spectacles, as they came in contact with the air expired, were covered with vapor. As the patient appeared to suffer from our presence, we went away.

"We made our third visit at two o'clock. There were still fifteen minutes before the beginning of the ecstatic crisis, which always took place punctually at a quarter-past two and ended at about half-past four. The pupils at this time were slightly contracted, the eyelids were almost entirely closed; the eyes, looking at nothing, were veiled from our view. We tried in vain to attract her attention; her mind was otherwise engaged, and her pains were evidently becoming more intense. At exactly a quarter-past two her eyes became fixed in a direction above and to the right. The ecstasy had begun.

"The time had now come to introduce those who were prompted by curiosity. This could now be done without inconvenience, for the ecstatic, for the ensuing two hours, would be lost to the appreciation of what might be passing around her. The room crowded could hold about ten persons, but enough were allowed to enter to make the total twenty-five. These placed themselves in two ranks, of which the front one kneeling allowed the rear one to see all that was going on. All this was done under the direction M. le Curé, who took every pains to give us a good view of what was going to happen.

"Louise was seated on the edge of her chair; her body, inclined forward, seemed to wish to follow the direction of her eyes, which did not look, but were fixed on vacancy. Her eyes were opened to their fullest extent, of a dull, lustreless appearance, turned above and to the right, and of an absolute immobility. A few workings of the lids were now observed and became more frequent if the eyelids were touched. The pupils, largely dilated, showed very little sensibility to light, and all that remained of vision was shown by slight winking when the hand was suddenly brought close to the eyes. The whole face lacked expression. At certain moments, either spontaneously or as a consequence of divers provocations, a light smile, to which the muscles of the face generally did not contribute, wandered over her lips. Then the face resumed its primitive expression, and thus she remained for the half-hour which constituted the 'first station.'

"The 'second station' was that of genuflection. It had failed at one time, but had again appeared. The young girl fell on her knees, clasped her hands, and remained for about a quarter of an hour in the attitude of contemplation. Then she arose and again resumed her sitting posture.

"The 'third station' began at three o'clock. Louise inclined herself a little forward, raised her body slowly, and then extended herself at full length, face downward, on the floor. There was neither rigidity nor extreme precipitation; nothing, in fact, calculated to produce injuries. The knees first supported her body, then it rested on these and the elbows, and finally her face was brought in actual close contact with the tiled floor. At first the head rested on the left arm, but very soon the patient made a quick and sudden movement, and the arms were extended from the body in the form of a cross. At the same time the feet were brought together so that the dorsum of the right was in contact with the sole of the left foot. This position did not vary for an hour and a half. When the end of the crisis approached the arms were brought close to the sides of the body, then suddenly the poor girl rose to her knees, her face turns to the wall, her cheeks become colored, her eyes have regained their expression, her countenance expands, and the ecstasy is at an end."

Further particulars are given, and an apparatus was constructed and applied to Louise's hand and arm so as to prevent any external excitation of the hæmorrhage. It was apparently shown that there was no such interference, for the blood began to flow at the usual time on Friday.

In addition to the stigmata and the paroxysms of ecstasy, Louise declared that she did not sleep, had eaten or drunk nothing for four years, had had no fecal evacuation for three years and a half, and that the urine was entirely suppressed.

M. Warlomont examined the blood and products of respiration chemically, and satisfied himself of their normal character, except that the former contained an excessive amount of white corpuscles.

When being closely interrogated, Louise admitted that, though she did not sleep, she had short periods of forgetfulness at night. On M. Warlomont suddenly opening a cupboard in her room, he found it to contain fruit and bread, and her chamber communicated directly with a yard at the back of the house. It was therefore perfectly possible for her to have slept, eaten, defecated, and urinated, without any one knowing that she did so.

The conclusions arrived at by M. Warlomont were, that the stigmatisations and ecstasies of Louise Lateau were real and to be explained upon well-known physiological and pathological principles, that she 'worked, and dispensed heat, that she lost every Friday a certain quantity of blood by the stigmata, that the air she expired contained the vapor of water and carbonic acid, that her weight had not materially altered since she had come under observation. She consumes carbon and it is not from her own body that she gets it. Where does she get it from? Physiology answers, 'She eats.'"

MM. Mauriac and Verdalle[1] give a very interesting account of an ecstatic woman who daily enacted the passion of Jesus, terminating in the usual manner with the representation of the crucifixion. This woman, Beguille, was of nervous temperament, had had many visions of the Virgin and of angels, and was accustomed to prophesy.

---

[1] "Étude médicale sur l'extatique de Fontet," Paris, 1875.

When visited by MM. Mauriac and Verdalle, Berguille was lying in bed. She is described as a woman of about forty-five years old, brown complexion, muscles and limbs well developed but without much fat, eyes blue, widely open, and staring vaguely. She smiled kindly when questions were put to her, and answered with sufficient intelligence.

On being asked why she was in bed, she answered that she was in pain night and day; and, when requested to state where she felt the most pain, she answered, the backs and palms of the hands, the tips and soles of the feet, and the right side. (It will be remembered that Louise Lateau had her pain and hæmorrhage in the left side, a difference which the miracle-believers ought to find it difficult to reconcile.)

Relative to her visions and what she heard during her ecstasies, she said that she saw Jesus Christ in his passion, that she heard voices, but she could not repeat what was told her. Her pulse was from 68 to 72.

At about one o'clock the ecstasy began. Her pulse rose to 80. She clasped her hands on her heart, her gaze became fixed, her eyes were widely opened, her lips moved as if she were murmuring prayers, and there were frequent movements of deglutition. Her pupils were slightly dilated, but contracted when a light was brought to them. Her limbs were rigid, but it was noticed that she flexed them very readily when she altered her position a little or arranged her dress. In a few minutes she raised herself somewhat awkwardly on her knees, her hands still being clasped and her eyes fixed. Then began the passion or the way to the cross, during which she walked on her knees around the bed, changing her position twelve times, and falling three times in the traditional manner. To make this journey required thirty-six minutes, and, this done, the next act, the crucifixion, was in order.

Suddenly she threw herself back on the bed, extended her arms from each side, and remained immovable. The pulse was 112, the respirations 100. The muscles of the chest seemed to be paralyzed, only the diaphragm acting. The eyes were closed.

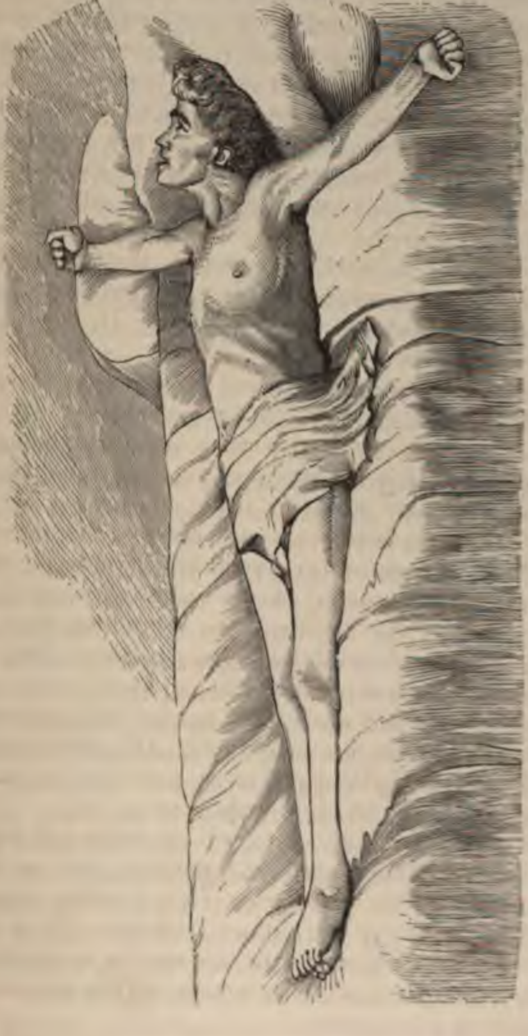
The limbs were in a state of forced extension and very rigid; the cutaneous sensibility to pinchings, prickings, and to the electrical stimulus was abolished. The latter, a very strong induced current, caused muscular contractions but no sensation. There was not the least flinching. Things went on in this way for over three hours, and then she sang the *Salve Regina*, exclaimed "Oh, what sorrow!" and gradually recovered her senses.

M. Bourneville[1] cites the case of Ler., a hystero-epileptic, to whom reference will be again made, who at one time had a cruciform paroxysm. Her head was strongly thrown back; her eyelids, half open,

---

[1] "Louise Lateau," etc., Paris, 1875, p. 13.

Fig. 103.



were in continual motion; the muscles of the jaws were contracted, and the muscles of the neck were hard and tense.

. The superior extremities were extended at right angles from the trunk, the hands closed, and the fingers flexed so strongly on the palms as to render it impossible to open them.

The inferior extremities were stretched out to their full length, the sole of one foot in contact with the dorsum of the other.

In a word, the rigidity was such that the body could have been raised from either end like a bar of iron (Fig. 103).

The attack lasted about four hours; then Ler. opened her eyes and recovered consciousness, exclaiming, "O my God, I was so happy!"

Two other interesting cases are described by M. Billet,[1] but the foregoing are sufficient to give the reader some idea of ecstasy as it appears in Catholic countries.

But the phenomena exhibited by Protestant ecstatics have been and are to this day fully as remarkable pathologically as those just described. Calmeil,[2] speaking of the Protestant theomaniacs of Languedoc and Cévennes, says:

"In general, they gave the name of *ecstatic period* to the agitation and improvisation which characterized the attack. All the inspired were fully persuaded that the Holy Spirit had entered into their breasts at the moment when they felt themselves constrained by an overwhelming power to prophesy. All expressed themselves as if the Spirit of God spoke to them the words they uttered."

Elizabeth Barton, called the "Holy Woman of Kent," announced, during an attack of hysteria, that a child then sick with a brain-fever would die. As she predicted, the event took place soon afterward, and the fulfillment of this prophecy at once gave the holy woman a great reputation. On this she announced that she was illumined by the Holy Spirit. She had numerous ecstatic paroxysms, during which she, according to her own account, was transported to heaven, and on her emergence she sang hymns, prayed, and made many predictions which astonished her admirers. At last, in obedience to an asserted command of the Virgin, she renounced Protestantism and took the veil. She continued to prophesy, and, growing bold, she predicted the speedy death of the king for his putting aside his wife Catharine of Aragon for Anne Boleyn. Henry VIII. was not of the temper to submit to this sort of holiness, so he had Elizabeth Barton beheaded as a pestilent woman, who was better out of the world than in it.

An ecstatic, in a paroxysm of rapture, having lost his speech, thus describes his regaining the faculty:[3]

"At length, after nine months of sobs and convulsions without speech, one Sunday morning as I was praying in my father's house, I fell into an extraordinary ecstasy, and God opened my mouth. During the ensuing three days I was constantly under the operation of the Holy Spirit, neither eating, drinking, nor sleeping, and I spoke often with more or less power, according to the nature of things. All in the family were convinced, as well by the extraordinary state in which they now saw me, as by the wonderful fasting of three days, during which I felt neither hunger nor thirst, that it was surely by the Sovereign Power that such astonishing things were done."

---

[1] "Contributions à l'étude des névroses extraordinaires," Paris, 1874.

[2] "De la folie," etc., tome ii., Paris, 1845, p. 288.

[3] Calmeil, *op. cit.*, p. 289.

In our own day, instances of ecstatic trance during camp-meetings, revivals, and the like, are common enough, and the number is greatly increased by spiritualism, mesmerism, and such like absurdities.[1]

As we have seen, many ecstatics pretend that they do not eat. Cases of the kind are reported very often, and have been noticed from an early period. Thus Schenckius[2] quotes from Paulus Lentulus the "Wonderful History of the Fasting of Appolonia Schreira, a Virgin, in Bern," in which it is stated that, being carefully watched by the orders of the magistrates of Bern, it was ascertained that there was no fraud, and she was dismissed as a genuine case of ability to live without food. During the first year of her fasting she scarcely slept, and in the second year not at all.

Another, and still earlier case, was that of Margaret Weiss, aged ten years, who lived in Rode, a small village near Spires, and whose history is given by Gerardus Bucoldianus.[3] Margaret is reported to have abstained from all food and drink for three years, in the mean time growing, walking about, laughing and talking, like other children of her age. She, however, during the first year suffered greatly from pains in her head and abdomen, and, *all four of her limbs were contracted.* She passed neither urine nor fæces. Margaret played her part so well that, after being watched by the priest of the parish, and Dr. Bucoldianus, she was considered free from all juggling, and was sent home to her friends by order of the king, "not without great admiration and princely gifts." The circumstances seem to have somewhat staggered Dr. Bucoldianus, for he asks, "Whence comes the animal heat, since she neither eats nor drinks, and why does the body grow when nothing goes into it?"

Of the cases that have been recently reported, that of the so-called Welsh fasting-girl[4] is one of the most remarkable, and a few years ago an account of its tragical ending excited a good deal of comment in the medical journals of Great Britain. Like the others, this was a case of hysteroid disease, and when she was so strictly watched that deception was no longer possible, she died in a few days of starvation. The startling heading to an editorial notice in the *Lancet*[1]—"Starved to Death"—expressed no more than the actual truth.

---

[1] For a full discussion of this subject, the reader is referred to the author's work "On Certain Conditions of Nervous Derangement," New York, 1881.

[2] "*Παρατηρήσεων*, sive observationum medicarum, rararum, novarum, admirabilium et monstrosarum volumen, tomis septem de toto homine institutum," Lugduni, 1606, p. 306.

[3] "De Puella quæ sine cibo et potu vitam transigit," Parisiis, ann. MDXLII.

[4] "A Complete History of the Welsh Fasting Girl, with Comments thereon, and Observations on Death from Starvation," by Robert Fowler, London, 1871.

In regard to the rarity of defecation and urination in cases of hysteroid disease there is no doubt. Such cases are often accompanied with vomiting, and then the matter ejected from the stomach contains urea and sometimes even fecal matter. A lady, not long since under my charge, in whom there were no other very decided hysterical symptoms, had an operation from her bowels never more frequently than once a month, and generally not so often. Every time she ate anything she vomited soon afterward, and the vomited matter always contained urea. She urinated about a tablespoonful every eight or ten days. The vomiting of fecal matter in cases of hysteroid disease is not so common. Briquet[2] reports a case as occurring in his own experience, in which there was no doubt that substances administered as enemas were vomited a few minutes afterward. Among other experiments, and in order to remove all doubt arising from the use of household substances, an injection of tincture of litmus was given immediately after it was brought from the pharmacy. The patient was told that coffee was to be injected. Twelve minutes afterward the tincture of litmus was vomited, its blue color turned to a red through the action of the gastric juice.

Less authentic, perhaps, is the following from Henricus ab Heeres:[3]

"A certain gentleman has lived several years without having had any operation from his bowels. About the middle of the day he sits down to his dinner, usually inviting several noble persons to eat with him. In an hour he rises from the table, after having eaten and drunk to his satisfaction, and retiring, vomits the dinner he had eaten the day before, but retaining all the dinner he has just taken. It is ejected putrid and filthy, differing in no respect from other excrement. He vomits with ease, and at once, throwing up the contents of the stomach which have remained from the previous day. Then he washes his mouth with clean water and returns to his friends to finish his repast. He eats no supper or breakfast, and thus he has done for about twenty years."

Stigmata, as occasional symptoms of the hysteroid condition, are well known to dermatologists, many cases of bloody sweat having been noticed. Those observed in the case of Louise Lateau were well studied by M. Worlomont, and they were found to differ in no essential respect from those previously observed, except in regard to the periodicity of the hæmorrhages—a circumstance, however, easily accounted for by the fact that the stages of excitement were regular. Hæmidosis, or bloody sweat, is to be regarded as one of the neuroses of the skin. An interesting case is reported by Wilson.[1] Mason Good cites authorities to show that it has taken place during coition, violent terror, and great bodily agony. Its occurrence in the hands, feet, and side, is to be explained by the fact that the attention is strongly concentrated on these parts, and it is in all probability kept in these situations by manual irritation. It is by no means certain that this latter was not the case with Louise Lateau, for M. Warlomont's apparatus was not of such a character as to prevent such action.

---

[1] *Lancet*, December 25, 1869.

[2] "Traité clinique et thérapeutique de l'hysterie," Paris, 1859, p. 316.

[3] "Observationes medicæ," Lipsiæ, 1645, lib. 1, ob. 29.

Causes.—Ecstasy, though not entirely confined to the female sex, is very much more common in women than in men. It appears to be produced in those who are of delicate and sensitive nervous organizations by intense mental concentration on some one particular subject—generally, one connected with religion, or some other abstract train of thought. It was formerly quite common among the inmates of convents, and is now not unfrequently met with at camp-meetings and spiritualistic gatherings.

There are no points about the **Diagnosis** requiring special consideration, and the **Prognosis** is always favorable, if the subject can be submitted to proper moral and physical treatment. As the disease is never fatal *per se*, we know nothing of its **Morbid Anatomy.** The pathology, as indicated by the symptoms, points to the implication of both the brain and spinal cord, but there is no satisfactory theory of the disorder other than that which refers it to cerebral and spinal preoccupation—a kind of setting of the current in one direction, whereby all other occupation is for the time prevented.

Treatment.—The means of treatment, though not differing essentially from those proper for catalepsy, require, nevertheless, special mention of some particulars. The influence of moral force in preventing and curing ecstasy is well marked, and many instances are on record in which epidemics of it have been arrested by arguments addressed to the fears of the subjects. I have several times aborted and prevented ecstatic manifestations by making preparations to cauterize the region of the spine with a red-hot iron.

A great deal can be done by giving as little notoriety to ecstatics as possible. They glory in the idea that they are of sufficient importance to excite attention and discussion, and they are accordingly stimulated to continue their performances so long as they are noticed and an air of mystery is attached to them.

Removal from all associations calculated to continue the exciting and morbid train of thought which has developed the disease under notice, should, of course, be a point in the treatment.

---

[1] "On Diseases of the Skin," American edition, Philadelphia, 1863, p. 551.

Electricity, and the other measures of treatment recommended for catalepsy, will prove serviceable in ecstasy. By galvanization of the sympathetic nerve, I, on one occasion, immediately cut short a paroxysm of ecstasy, and, by continuing the practice every alternate day for about six weeks, effectually cured the patient, who for several years had been subject to seizures every two or three days.

As means for improving the general health are almost invariably required, iron, quinine, and strychnia, in the combination recommended on page 54, may be administered with advantage. I have great confidence in the bromides, and the patient should be to a moderate extent brought as soon as possible under the influence of some one of those previously mentioned.

III.

HYSTERO-EPILEPSY.

The combination of hysteria with epilepsy has long been recognized as existing and as giving rise to one of the most frightful affections to be found in the whole range of neurological medicine. In the present state of our knowledge it would, perhaps, be going too far were we to pronounce positively in favor of its being a distinct pathological entity with a different anatomical substratum from either hysteria or epilepsy, and yet the phenomena are so distinct that we certainly are warranted in considering it separately from either of these diseases.

Symptoms.—An attack of hystero-epilepsy is characterized by the occurrence of convulsions more or less resembling those of epilepsy. There is usually in the first place a well-marked tetaniform spasm, though, sometimes, this is not very decided, and occasionally is not observed at all. Then follow clonic convulsions, during which the patient froths at the mouth and may pass the urine or bite the tongue, though these phenomena, especially the latter, are rare. Loss of consciousness exists during this stage.

Next ensues a remarkable series of movements, at the beginning of which, or during their continuance, the patient recovers consciousness to such an extent as to answer questions, although there is no after-recollection of the incidents that may have occurred. These movements are apparently voluntary, and consist of the most extraordinary contortions of the muscles of the face, neck, trunk, and extremities, so that superstitious people might well imagine the existence of an internal or external diabolical agency. During the continuance of this part of the paroxysm the patient tears with the hands and teeth any thing tearable that comes within reach, and continually utters inarticulate sounds or words apparently in relation with the ideas passing through the mind. Finally, the purely hysterical element ceases to predominate, and the patient alternately weeps and laughs, and gradually acquires a knowledge of what is passing around.

During the whole of the paroxysm the face is flushed, the pupils are moderately contracted, the pulse is accelerated, the perspiration is increased in quantity, and the respiration is hurried and irregular.

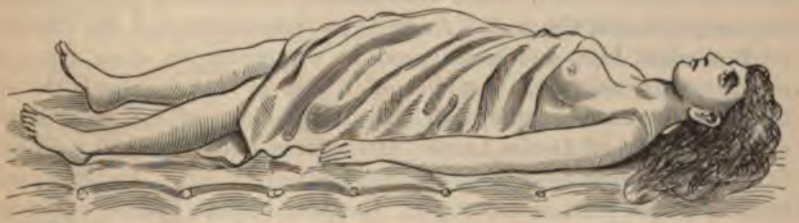
But there are numerous deviations from this type of a seizure. Sometimes the tetanic spasm is wanting, and again it, or some modification of it, may constitute the most marked part of the convulsive period. Thus in a lady, who was lately under my charge, the paroxysm began with an opisthotonos, which was immediately relaxed, and again renewed, to be again relaxed, and so on, for over half an hour.

In a woman whom I saw in the Pennsylvania Hospital several years ago, in the service of Dr. Pepper, the convulsions consisted of a series of rapid movements produced, as the patient lay on her bed, by the bending back of the body, so as to throw it into an opisthotonotic position, the head and heels alone touching the bed, and then, the muscles being suddenly relaxed, allowing the buttocks to fall with force on the bed. These actions were continued with great rapidity, and without intermission for an hour or more, and were succeeded by a period during which there were alternate laughing and weeping.

Such cases are what Sauvages designated as *hysteria libidinosa*.

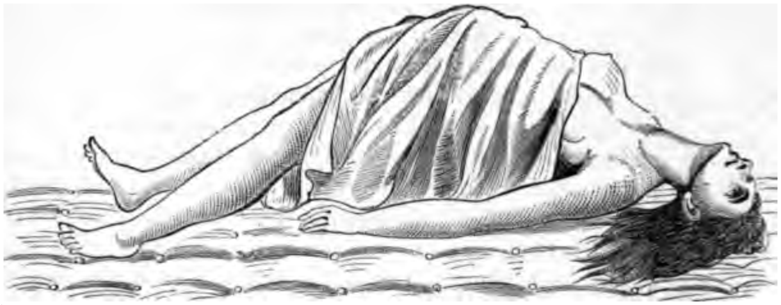
But, in a case now under my care, the patient, a woman, has daily attacks at about the same hour—three o'clock P. M.—which are more distinctly tetaniform in the beginning than any that have come under my observation. They consist of a series of opisthotonotic spasms, during which the body is extremely rigid. The convulsion is, however, unlike the others referred to, very slowly developed. The body extended at full length in the recumbent posture gradually becomes rigid, the legs are slightly abducted, the arms are pressed strongly against the sides, the jaws are tightly closed, and the gaze fixed (Fig. 104). Respiration is entirely suspended, and the heart beats rapidly, sometimes as frequently as one hundred and sixty per minute. Then the body is slowly bowed, so that the head and heels alone touch the bed, and is so rigid and strongly arched that no ordinary force, such as a powerful man can exert, suffices to overcome the tonicity of the muscles. In about a minute from the beginning of the rigidity, the spasm suddenly relaxes, and with a long-drawn inspiration the paroxysm ends—to be again re-

FIG. 104.



sumed in a few minutes with a like sequence. In the accompanying woodcut (Fig. 105) is an exact representation of this patient when the tetanic spasm is at its height.

FIG. 105.



In this case there is a distinct aura starting from the left ovary, and strong pressure exerted upon this organ suffices generally, though not always, to cut short the series of paroxysms.

Under the name of demonomania many cases of hystero-epilepsy have been described, and the disease, like chorea, has at times prevailed epidemically. At Loudun, in France, it led to the death at the stake of Urbain Grandier, the nuns, who were its subjects, accusing him in their delirium of having bewitched them. At Marseilles, Father Louis Gaufridi, a man of education and of strict morality, was accused by two Ursuline nuns of having debauched them through diabolical agency. At the time of the accusation, these nuns, one of them only nineteen years old, were suffering from attacks characterized by hallucinations and illusions, fearful epileptiform and cataleptiform convulsions, and delirious ravings—all of which were ascribed to the devil moved and instigated by Louis Gaufridi. At first, the accused denied the charges made against him, and endeavored by arguments to show the real nature of the seizures. But the effort was in vain, just as is the attempt now to convince the credulous and ignorant of the real nature of the seizures of Louise Lateau, Bernadette Soubirous—who evoked Our Lady of Lourdes—and of the hundreds of mediums, ecstatics, and hysterics, who pervade the world. Gaufridi became insane, and confessed all that was laid to his charge, with numerous other offenses which had not been imagined. He declared that he had worshiped the devil for fourteen years; that the demon had given him power to render amorous of his person all women on whom he should breathe, and that he had thus overcome several thousand women! Gaufridi, after horrible tortures, was burned at the stake; and the two nuns "continued to be delirious," as well they might.

As showing the nature of the phenomena exhibited in cases of demoniacal possession and their resemblance to the symptoms of hystero-epilepsy and other forms of hysteria, I subjoin the following questions as proposed by Santerre, priest and promoter of the diocese of Nîmes, to the University of Montpellier:

Question 1. Whether the bending, moving, and removing of the body, the head touching sometimes the soles of the feet (opisthotonos), and other contortions and strange postures, are a good sign of possession?

2. Whether the quickness of the motion of the head forward and backward, bringing it to the back and breast, be an infallible mark of possession?

3. Whether a sudden swelling of the tongue, the throat, and the face, and the sudden alteration of the color, are certain marks of possession?

4. Whether dullness and senselessness or the privation of sense, even to be pinched and pricked without complaining, without stirring, and even without changing color, are certain marks of possession?

5. Whether the immobility of all the body, which happens to the pretended possessed by the command of their exorcists, during and in the middle of the strongest agitations, is a certain sign of a truly diabolical possession?

6. Whether the yelping or barking like that of a dog, in the breast rather than in the throat, is a mark of possession?

7. Whether a fixed, steady look upon some object, without moving the eye on either side, be a good mark of possession?

8. Whether the answers that the pretended possessed make in French to some questions that are put to them in Latin are a good mark of possession.

9. Whether to vomit such things as people have swallowed be a sign of possession?

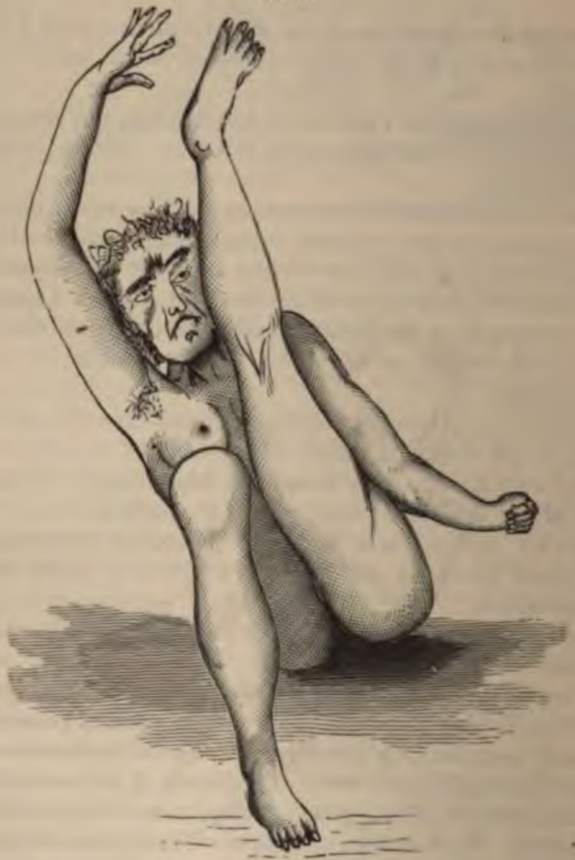
10. Whether the prickings of a lancet upon divers parts of the body without blood issuing therefrom are a certain mark of possession?

All these questions, to the credit of medical science—which has always, notwithstanding the weakness of some of its professors even in our own day, been steadily opposed to supernaturalism—were answered in the negative. No one can read them without being struck with the facts that Father Santerre was at least a good symptomatologist, and of the absolute identity of the phenomena cited, in all essential characteristics, with those which in our day are said to be of mystical origin, but which in reality are hysterical or hysteroid. We might reproach Father Santerre and his coadjutors more forcibly, if we had not ourselves killed witches and presided at the birth of spiritualism.

No one has written with greater effect in regard to the manifestations of hysteria and hystero-epilepsy than Charcot. As a most striking case of the latter affection, I cite from him the following instance[1] already referred to in another connection under the head of ecstasy.

Ler., aged forty-eight years, is a patient well known to all physicians who visit the Salpêtrière as one of the most remarkable instances extant of hystero-epilepsy. Her menstruation has ceased for four years and yet all the neurotic symptoms persist. She is a demoniac, a possessed, and presents a striking example of that type of hysteria manifested by the "Jerkers" in "Methodist camp-meetings," and who exhibit in their paroxysms the most frightful attitudes.

FIG. 106.



The probable origin of these nervous phenomena in Ler. deserves to be noted. She has had, as she says, a series of frights. At eleven years of age she was terrified by a furious dog. At sixteen she was frightened at the sight of the corpse of an assassinated woman, and again about the same time, when going through a wood, by robbers who attacked her and took her money.

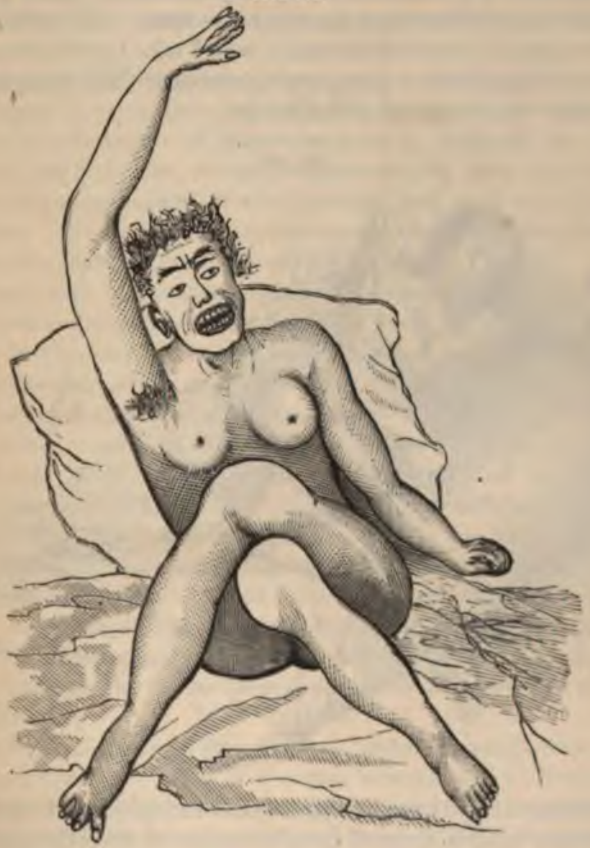
---

[1] "Leçons sur les maladies du système nerveux faites à la Salpêtrière," Paris, 1872–'73, p. 301, *et seq.*

With her there are local hysterical manifestations consisting of hemi-anæsthesia, ovarian tenderness, paresis, and at times contraction of the limbs on the right side. Sometimes these symptoms are shown on the left side also.

The attacks, which are announced by a well-marked ovarian aura, are characterized at first by epileptiform and tetaniform convulsions; after which come extensive movements of an intentional character, in which the patient assumes the most hideous postures, recalling the attitudes which history ascribes to demoniacal possession (Figs. 106 and 107). At the moment of the attack she is seized with delirium, which evidently turns on the events which have produced the initial seizures.

Fig. 107.



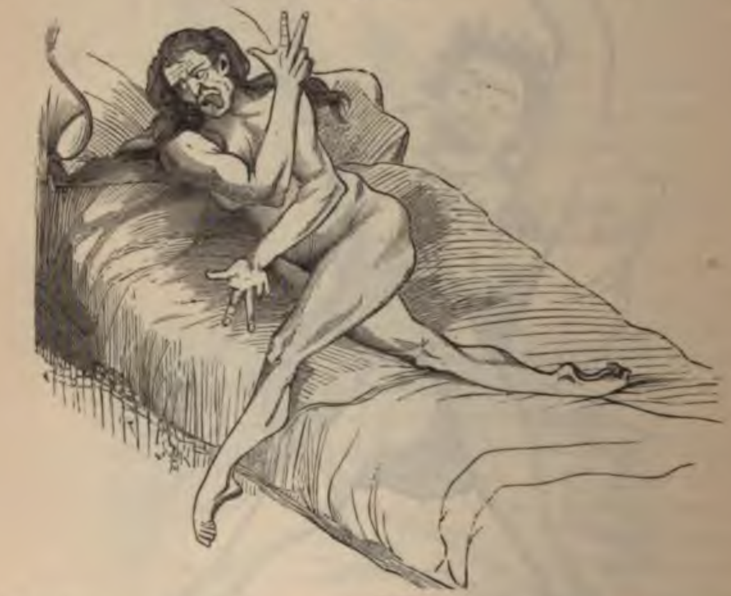
She hurls invectives at imaginary persons. "Scoundrels! robbers! brigands. Fire, fire! Oh, the dogs, they bite me!"

When the convulsive part of the accession is over, there ensue, generally, hallucinations of sight—she sees frightful animals, skeletons, and spectres; a paralysis of the bladder; a paralysis of the pharynx; and a contraction, more or less permanent, of the tongue.

It is therefore necessary for several days to feed her through a tube, and to empty the bladder with a catheter.

Later, M. Bourneville[1] has given an account of Ler., somewhat fuller than that of M. Charcot, to which, as showing how Ler. had at one time exhibited phenomena of ecstasy similar to those present in Louise Lateau, reference has already been made. In further illustration of the period of contortions in her case I take from M. Bourneville's excellent monograph the accompanying woodcut (Fig. 108), made from a sketch taken on the spot by M. Charcot.

FIG. 108.



In the intervals between the paroxysms the subjects of hystero-epilepsy generally exhibit some of the phenomena of hysteria such as hemianæsthetic contractions, ovarian tenderness, paralyses, etc.

Relative to the **Causes**, the **Prognosis, Diagnosis, Morbid Anatomy and Pathology**, and **Treatment**, there is nothing to add to the remarks already made when hysteria, catalepsy, and ecstasy, were under consideration.

---

[1] "Louise Lateau, ou la stigmatisée belge," Paris, 1875, p. 38, *et seq.*

CHAPTER VII.

MULTIPLE CEREBRO-SPINAL SCLEROSIS.

We have already considered the subject of sclerosis as it affects the brain and spinal cord separately. We have still to treat of it as existing in these nervous centres simultaneously. Although recognized, over thirty-five years ago, by Cruveilhier and Carswell, it is only recently, mainly through the observations of Charcot and Vulpian, that attention has been again directed to sclerosis of the cerebro-spinal variety, a form which differs from those already described in this treatise, both in its extent and in the symptoms by which it is characterized.

Symptoms.—The initial symptoms vary according as the morbid process begins in the brain or spinal cord. In the former case, the first prominent manifestation of disease may be an epileptic fit. In other cases, there are headache, vertigo, ocular troubles, such as ptosis, diplopia, or amblyopia, failure of the hearing, and, very often, defective articulation. The mind does not participate to any considerable extent, unless the hemispheres be involved in the lesion.

Or, there may be hemiplegia as a consequence of cerebral congestion, and even mania, from a like cause. These attacks are sometimes frequent, and usually leave more or less mental weakness after them.

Tremor is often first seen in the tongue, more frequently in the eyeball, of one or both sides, which oscillates when the patient is told to turn it inward or outward, but which is steady when he looks directly to the front. This tremor is called nystagmus, and is, as we have already seen, met with in other diseases of the nervous system. According to Ferrier's[1] observations, it is due to lesion of the cerebellum, and when met with in the disease under notice points to this organ as one of the seats of the morbid process. In the case of a woman who attended my clinic at the Bellevue Hospital Medical College, nystagmus was the only symptom observed for over a year, and then gradually other phenomena of the cerebro-spinal form of sclerosis made their appearance.

Tremor is indicative of loss of power, and it gradually becomes more strongly marked and extends to other muscles of the body as other parts of the cerebro-spinal system become involved. It is never, however, a constant phenomenon in any form of sclerosis affecting the spinal cord alone. Its presence is peculiar either to cerebral disease or to lesions occurring at the same time in the brain and spinal cord.

After a time, which is subject to great variation in different cases, the loss of power extends to the limbs, and this feature is often accompanied with aberrations of sensibility. If, as is generally the case, the membranes of the cord are congested or inflamed, there are spasmodic jerkings or twitchings of the limbs, but in some cases these are never observed. In the case of a gentleman from South Carolina, who consulted me at the instance of my friend and colleague Prof. J. T. Darby, and who was obviously affected with multiple cerebro-spinal sclerosis, there had never been the slightest involuntary movement, independent of the peculiar form of tremor in the limbs which constitutes so prominent a feature of the disease.

---

[1] "Experimental Researches in Cerebral Physiology and Pathology," "West Riding Lunatic Asylum Medical Reports," vol. iii., 1873, p. 69.

The lower extremities are generally very much more paralyzed than the upper, and, when they become involved, festination often makes its appearance. The gait of the patient, thus, becomes similar to that of a person in whom the lesion is limited to the brain.

If the sclerosis begins in the brain before attacking the spinal cord, tremor precedes the paralysis—the affection being then entirely cerebral in character; but, when, as is generally the case, the lesion appears primarily in the spinal cord, paralysis is noticed before the tremor. In fact, there is never, as previously insisted on in my remarks on multiple cerebral sclerosis, any tremor, unless the superior ganglia of the cerebro-spinal system are involved. The fact that it is only shown when a voluntary movement is made also assists us to distinguish it from the tremor of multiple cerebral sclerosis, as well as from that of paralysis agitans. In the cerebro-spinal form of the disease, therefore, the patient remains without tremor so long as he is quiescent. But, if he attempts to cross one leg over the other, or to carry a glass of water to his lips, the extremity executing the movement is at once seized with tremor, and the act is performed with great difficulty.

The ability to place the fingers on any part of the body, unassisted by the eyesight, is impaired, as in the cerebral form of the disease, and in sclerosis affecting the posterior columns of the spinal cord.

As the disease advances, the paralysis becomes more strongly marked; the limbs are permanently contracted; the bladder loses its expulsive force; its sphincter no longer completely closes the orifice; the bowels become obstinately constipated, and there is a strong tendency developed to the formation of bed-sores. The head-symptoms likewise increase in intensity, but the mind remains clear to the last in the great majority of cases. Indeed, my observation of many cases has convinced me that in the cerebro-spinal form of sclerosis the hemispheres are not often involved, even when the disease has lasted several years.

The difficulties of articulation notably increase, and the muscles of deglutition likewise become involved. In consequence, the saliva is not swallowed as often as it should be, and it therefore dribbles from the mouth. Mastication is difficult, and the facial muscles gradually become involved. The countenance of the patient at this period is not unlike that of a person suffering from glosso-labio-laryngeal paralysis, as in fact might be expected, the same nerves and muscles being involved. Finally, the patient dies from exhaustion, or from some intercurrent disease.

Few diseases are so irregular and ununiform in their phenomena as the cerebro-spinal form of sclerosis. This is due to the fact that the organs liable to be the seat of the disease are numerous and of varied functions. The essential feature of the affection is tremor occurring generally after paralysis, and only manifested during the performance of voluntary movements. It is not always necessary, however, that the movements should be of the partially-paralyzed limbs, for I have seen cases in which tremor was excited in a paretic leg by the act of executing voluntary movements with a sound hand.

The following histories will contribute to a fuller understanding of the subject :

Cruveilhier[1] reports the case of a cook, aged thirty-seven, who six years before coming under observation noticed that he was losing power in the left leg, so that he nearly fell in the street. Three months subsequently the right leg became similarly affected, and then the superior extremities followed. They were tremulous and weak, but the patient was still able to use them to some extent. The sensibility remained intact, and the reflex faculty of the cord was unimpaired. In other respects the patient was condemned to immobility. There were no spasmodic retractions of the limbs, and no painful contractions. The articulation was imperfect, but the intelligence was unaffected. There appear to have been no marked head-symptoms in this case. "Point de cephalalgie, jamais de cephalalgie, le malade entendait à merveille." After death there was found gray degeneration of the spinal cord, of the medulla oblongata, of the pons Varolii, of the right cerebral peduncle, of the right optic thalamus, of the corpora callosa, and of the fornix. The hemispheres were not involved.

Two other cases, similar in general character to the foregoing, are given, in neither of which were the hemispheres involved.

Another case, that of Josephine Pajet, is cited by Cruveilhier.[2] In this there was almost complete insensibility of the inferior extremities, though the patient was able to move the toes, the feet, and the legs. There were no cramps and no contractions. There was also diminished sensibility of the superior extremities. All the limbs were weak, and the arms were affected with tremor. The patient could walk and sew when first seen. The right hand was stronger than the left. There was a sensation of a tight band around the abdomen. After death there was gray degeneration of the cord, and of the pons Varolii.

---

[1] "Anatomie pathologique du corps humain," Paris, 1835, 1842, tome ii., liv. xxxii., Fig. 4, Pl. 2.

[2] *Op. cit.*, liv. xxxviii., Fig. 1, Pl. 5.

In none of these cases were there spasmodic jerkings or tonic contractions of the limbs. Two cases have been reported by Friedreich.[1] In one of these a man, aged twenty-one, was the subject. Among the first symptoms were mental excitement, vertigo, pain in the head, and weakness of the lower extremities. The gait was unsteady, and there was tremor upon any emotional excitement, or on the attempt to execute movements. This affected the upper and lower extremities, the head, and the eyeballs. After death, patches of sclerosed tissue were found on the tubercula mammillaria, the cerebral peduncles, the pons Varolii, and the medulla oblongata.

The other case was that of a woman, aged twenty, who was attacked, when seventeen years of age, with weakness of the right leg. Soon afterward the left became affected, and subsequently the arms. These latter were rendered tremulous at every attempt to move them. The speech was implicated, and there was nystagmus. The mind was weakened, and the sensibility was impaired.

In the first of these cases the disease appears to have begun in the brain; in the second in the spinal cord.

Vulpian,[2] under a title which goes to show how even the best authorities have confused the whole subject of sclerosis, describes an interesting case communicated by Charcot. In this instance a woman, aged forty-three, of nervous temperament, had been subject to frequent attacks of facial neuralgia, and had often suffered from vague pains without determinate seat. In 1856, she suffered from attacks of vertigo, which, from being rare at first, subsequently came on five or six times a day. Sometimes she fell, but never lost consciousness, or had any convulsive movement.

Shortly afterward, during the night, she was seized with vomiting, cramps in her limbs, and a numbness of the right side. In the morning she was hemiplegic. Fifteen days afterward motion reappeared in the arm, but the leg remained paralyzed. In 1859, she had another attack of hemiplegia, and this time was deprived of speech for fifteen days. After this seizure, there were contractions of the flexors of the fingers, and of the forearm of the right side. In 1861, she had a third attack.

In 1862 (January 1st), she came under M. Charcot's care.

The intellectual faculties were not involved. The right superior extremity was almost entirely paralyzed, and was in a state of rigidity and contraction. The lower extremities were permanently extended, and could not be flexed but by great effort. Sensibility was perfect throughout, and reflex movements could still be excited. She died February 9th.

---

[1] "Deutsche Klinik," No. 14, 1856.

[2] "Note sur la sclérose en plaques de la moëlle épinière," *L'Union Médicale*, No. 70, Juin 14, 1866, p. 507. Like other writers, Vulpian, in this paper, brings together cases which have no affinity except as regards the general character of the lesion.

On post-mortem examination, patches of sclerosed tissue were found in the right middle cerebral peduncle, the pons Varolii, the medulla oblongata, and the cervical region of the spinal cord. The hemispheres were perfectly healthy.

In this case, it is probable that the contractions were mainly due to secondary degeneration of the cord, a condition which, as we have seen, is analogous to sclerosis. It will be observed that there were no tremors, either with or without voluntary motions.

Another important case has been reported by M. Magnan:[1]

A woman, aged thirty-four, came under observation in July, 1869. In 1848, when thirteen years of age, she had an attack of typhoid fever, from which she lost her sight. The first symptom of her disease occurred in 1867, and consisted of trembling of the hands and arms whenever she endeavored to execute any difficult movement. Before long, the tremor involved the lower extremities; but there was no paralysis till about eight months previous to her admission to the hospital. At this time, every effort at motion caused tremor. The hands, arms, legs, eyeballs, and even the muscles of the trunk, were involved. The articulation was defective, and there were various painful sensations in different parts of the body. Ophthalmoscopic examination showed atrophy of the optic disks and nerves.

The diagnosis in this case was multiple cerebro-spinal sclerosis—an opinion which I do not think is warranted by the facts. The lesion was probably entirely confined to the brain. The main reason which leads me to entertain this view is, that the tremor appeared before the paralysis. I cite the case for the purpose of showing how little accord there is among authors relative to the association of symptoms with lesions in the several forms of sclerosis.

Thirty-one cases of what the symptoms indicated to be the cerebro-spinal form of sclerosis have been under my care; and, though I have not had the opportunity of verifying my diagnosis in a single instance, I think the symptoms have been of such a character as to indicate the existence of the lesion so graphically described by Charcot, Friedreich, and Bourneville and Guérard.[2] The fact, that several of the histories were written out before Charcot's investigations gave me a clew to their real import, will tend, I think, to increase their value.

Mr. M., a gentleman fifty-three years of age, consulted me April 8, 1865, at the instance of my friend Prof. Fordyce Barker, M. D., for partial paralysis with tremor, mainly affecting the right arm and leg. Two years previously he had suffered from vertigo and headache, which were followed by a slight attack of hemiplegia of the right side, unattended by loss of consciousness. He gradually recovered from this, but, about six months before he came under my observation, he noticed that his right leg began to drag, and, soon afterward, that the arm of the same side became weak. About the same time he had headache, vertigo, and weakness of sight. A short time subsequently—about a month as well as he could recollect—the arm was seized with tremor while attempting to carry a glass of wine to his lips. The agitation continued to grow more violent on any voluntary movement of the arm, and gradually his speech became involved.

---

[1] "Mémoires de la société de biologie," Paris, 1869.

[2] "De la sclérose en plaques disseminées," "Nouvelle étude sur quelques points de la sclérose en plaques disseminées," Bourneville, Paris, 1869.

When I saw him he was still suffering from occasional attacks of vertigo and headache; the lips were agitated whenever he attempted to move them, the tongue was tremulous, and his speech was consequently halting and jerking. There was also nystagmus, a symptom which he had not noticed.

The right arm was unaffected with tremor so long as he allowed it to rest on his knee or to hang by his side; but, in the act of moving it, the whole extremity was agitated by a series of short, vibratory motions, consisting of flexions and extensions, which continued so long as he persevered in the movement, or kept the arm in any position requiring muscular exertion. The right leg was weak, and dragged so that he struck his foot against any slight obstruction. There was a little tremor in it when he attempted to cross it over the other as he sat in a chair.

I treated him solely with the primary galvanic current, which I passed through the brain and spinal cord—the first time such an operation was performed in this country for the treatment of disease. My diagnosis was incipient softening of the ganglia at the base of the brain and of the upper portion of the spinal cord. My opinion was, that the hemispheres were not involved, as there were no symptoms indicating mental weakness or disturbance.

I made an application of about fifteen minutes' duration every day. He gradually but rapidly improved, and to such an extent that on the 19th of April he wrote to me as follows:

"Yesterday must be marked with a white stone as the best day yet. Foot active, hand and arm steady, and spirits good. If we can manage to fix these good effects, cure is certain.

"I hope the magic pile will be ready to repeat its good work on Saturday next."

He continued to improve for several weeks, then gradually went back to his former condition, and from that rapidly grew worse. The paralysis invaded the other side, then tremor followed, the speech became much more difficult, and he died in the country two years subsequently.

Miss H., of Connecticut, aged thirty-five, consulted me January 20, 1870, for paralysis and tremor. About two years previously, she had noticed a weakness of the right arm, which had been preceded by occasional attacks of not very severe headache and vertigo. The arm gradually became weaker, and in the course of a few months began to shake whenever she attempted to use it. Before the year had expired, the right leg began to drag a little, and lost a good deal of its natural strength. Her speech also became difficult, not from any failure to remember words, but from tremor of the tongue and weakness, with a little rigidity of the lips.

When I saw her, the articulation was halting and syllabic; there was nystagmus in both eyes; the right arm was very weak; she could only move the index of my dynamometer four degrees, equivalent to a pressure of two pounds and a half, while with the left hand she could move it twenty-eight degrees. Every attempt to move the arm caused trembling of the whole extremity. So long as she refrained from any exertion of voluntary power, it remained free from agitation. She could not write, owing to the tremor which the effort to do so excited. There was slight tremor in the leg, when she slowly raised the foot from the ground.

The mind was perfectly intact, and she was entirely free from any emotional weakness.

In this lady's case I diagnosticated multiple cerebro-spinal sclerosis —the "sclérose en plaques disseminées" of Charcot.

I treated her with the chloride of barium and the primary galvanic current. By the following autumn she had improved so much that she could walk several miles without fatigue, lifted her foot clear of the ground, could move the index of the dynamometer to thirty degrees, was free from tremor, except when she attempted to write, and then it was only manifested to a slight extent. I now ceased using the galvanism, but continued the chloride of barium. On the 28th of January, 1871, she paid me a visit. She was then walking well, but there was still a very slight tremor when she attempted to execute delicate or difficult movements with the right arm. I directed the continuance of the barium.

Mr. H., of South Carolina, a highly-educated and intelligent gentleman, consulted me, September 12, 1870, for paralysis and tremor. As he entered my consulting-room, the tendency to festination was exceedingly well marked. On examination, I found his mind perfectly clear. There were nystagmus and syllabic articulation. On moving the left arm or left or right leg, the limb became tremulous. There had never been any head-symptoms.

On the 19th, at my request, he wrote a short account of his disease, which I here transcribe:

"I was never robust in health, but, on the other hand, I have never had, since childhood, a serious spell of sickness. My manner of life has been sedentary—that of a student. I was always careful not to overtask myself until I became engaged, in the year 1864, in a mathematical research. I was for a considerable length of time very much absorbed in this work, and allowed it to encroach seriously upon my hours of recreation and sleep.

"In the fall of 1865, after having accomplished the above work, I observed a slight lameness in my left foot—a tendency to strike the toe against the inequalities of the ground—an inability to raise quickly enough the front part of the foot.

"After my return home, summer of 1866, from Europe, where I had spent five or six years, the lameness in my foot increased rapidly, and in the winter of 1866–'67 a lameness in my left hand was very perceptible—an inability to move the fingers quickly, and a tremor, particularly of the thumb, when I attempted to do so.

"The above symptoms have gradually grown worse, and within the last year the right leg has become involved, to the extent that it begins to shake when I stand upon it, and it shakes even while sitting, when I am under excitement, or when I execute difficult voluntary motions with my hands.

"The disease seems to make greater progress in hot weather. I have at no time suffered pain, my appetite and digestion are good, and I generally sleep well."

This gentleman improved greatly through the use of the primary galvanic current, chloride of barium, and tincture of hyoscyamus, during the two weeks that he remained in New York under my care. On his return to South Carolina he took a primary-cell battery with him.

On the 11th of January, 1871, he wrote to me as follows:

"Sometimes I thought I was improving slowly, or at any rate not losing ground, and then again, for several days together, I would feel confident that I was falling back. But now I think I can certainly say I am growing worse. All my symptoms have been worse—lamer, more nervous, and the disease more general in its effects. My right hand, which has heretofore been comparatively unaffected, is now seriously implicated, and yet I still manage to write after a fashion. I find it very difficult to dress myself, and I must make several attempts before I can get up from a sitting or a lying posture.

"What could have caused the improvement that took place while I was under your immediate treatment?"

In this case I diagnosticated multiple cerebro-spinal sclerosis, and I think those acquainted with the disease will agree with me in my view of the case; and yet there was as strongly-marked festination as I have ever seen. The gentleman could trot well, could mount a staircase without much difficulty, but walking slowly, or descending stairs, troubled him greatly. According to some authors, this symptom would, of itself, have been sufficient to contraindicate the existence of sclerosis, and to have placed the disease among the neuroses. My views on this point have already been expressed under the head of multiple cerebral sclerosis.

J. F., a gentleman of this city, forty-two years of age, consulted me November 29, 1870. On the 4th of July previously he had indulged rather freely in champagne, and the following morning awoke with severe headache, vertigo, and nausea. Although he recovered from this attack, he never felt quite as well as before, and was frequently subject to headache and vertigo—symptomatic, as he thought, of gastric disorder. About a month after his first symptoms he was suddenly conscious of a singular sensation about his left eye, and on looking in the glass discovered that the upper lid had dropped, and that he could not raise it. This was about five o'clock in the afternoon, and by ten that night the lid entirely covered the pupil. The following morning it was not so low, but he found that he saw double. He continued to attribute all his troubles to the stomach, and began taking some quack remedy recommended to him for dyspepsia.

In the course of a few days, feeling no better, he went to the seashore, and while there noticed that his right arm became weak, and that he frequently let things drop from his hand. He had difficulty in shaving and in dressing himself, from inability to coördinate the muscles, and there was numbness of the ends of the fingers. During all this time he had suffered more or less from headache, vertigo, and double vision, and the ptosis still continued. Gradually the left arm became involved, and, by the time the paresis in this extremity was well established, the right arm was affected with tremor, but only when he attempted to execute movements with it. Thus, as he said, he could place the hand on a table and it would continue perfectly quiet ; but, as soon as he took a pen to write, or even endeavored to raise the hand from the table, it was seized with tremor. The left arm soon became similarly affected, and eventually the left leg lost strength and was rendered tremulous by any attempt at muscular exertion. He noticed also, what, as I afterward learned, his friends had perceived several weeks before, that his articulation was imperfect, and that is was necessary for him to make a mental effort to talk distinctly.

He returned to the city about the middle of October, and employed a "rubber" to restore, as he said, the circulation to his limbs. Continuing to get worse, he consulted me.

At this time there was festination. The speech was syllabic and accentuated, the tongue and lips were paretic and tremulous, there was nystagmus in both eyes, ptosis and diplopia from paralysis of the left sixth nerve, and dilated pupil of the right eye. There were also occasional headache and vertigo, but not to the same extent as at first.

Both arms and the left leg were partially paralyzed. He could not raise either upper extremity out from the side, owing to the complete paralysis of the deltoids, but he could flex both forearms, and move his hands and fingers tolerably well. There was no tremor while he refrained from using them, but the least attempt at voluntary motion excited them to agitation. The same was true of the left leg. Examination with the ophthalmoscope showed both optic disks to be white, and the retinal vessels small and straight.

With the dynamometer he could only exert a pressure of nine degrees with the right hand and eleven with the left. The line made with the dynamograph was descending, showing his inability to maintain, even for a short time, a uniform muscular contraction.

There was no loss of sensibility, except in the upper extremities. He had occasionally suffered from pains in the back, about the region of the shoulders.

The power over the sphincters was intact.

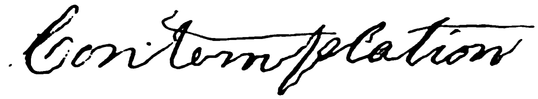
This gentleman could stand and walk as well with his eyes shut as with them open. On rising from his chair, which he did with difficulty, he always felt impelled to take a few steps forward, which were a stagger rather than a voluntary movement. In walking, the body was inclined forward, and he went in a kind of jog-trot.

He attributed his disease to dissipation of all kinds, in which opinion I expressed my concurrence.

Under treatment with galvanism, hyoscyamus, and chloride of barium, this patient has improved, but not as yet sufficiently to warrant any strong hope of a permanent cure.

A gentleman from the northern part of the State of New York consulted me in January, 1871, and again in March. His symptoms, though decided, were not very severe in character. Gradually, however, there had been for two years a loss of power supervening in the muscles of the right side of the body, and lately ocular troubles had made their appearance. Tremor, on making any voluntary movement, was just beginning to appear when I last saw him. Its influence over his handwriting is seen in the following *fac-simile:*

FIG. 109.


Contemplation

One patient, with multiple cerebro-spinal sclerosis, attends the outdoor department of the New York State Hospital for Diseases of the Nervous System. He has marked head-symptoms. And another, from Philadelphia, who was supposed to be suffering from cerebral disease, consulted me a few days ago. In this case the affection probably resulted from a fall.

The remaining cases do not present any such peculiar phenomena as to warrant their histories being given in detail.

Causes.—Nothing very definite is known of the etiology of the affection in question. It probably is induced by such causes as give rise to the purely cerebral form of the disease. Age does not, however, appear to exercise so important an influence. Eleven of my cases were over fifty years, and one of them, the gentleman from Philadelphia, was over sixty; seventeen were over forty and under fifty, and three were between thirty and forty. All were males but four.

In seven cases, it was apparently caused by excessive mental application, in two by anxiety, in one by a fall, in six by dissipation. In the remaining cases I could discover no obvious cause. In none of them was there a rheumatic, syphilitic, or other morbid diathesis.

Diagnosis.—The facts of the tremor making its appearance after the paralysis, and of its only—or, at least, with rare exceptions, and then only in the latter stages of the disease—being manifested when voluntary movements are being made, will suffice to distinguish the cerebro-spinal form of sclerosis from either of the other varieties. The points to recollect are these: that, in simple cerebral sclerosis, the tremor appears before the paralysis, and does not depend on the voluntary contraction of muscles for its excitation; in simple spinal sclerosis there is no tremor at all. I have already insisted on these distinctions in my remarks on the other forms of sclerosis of the nervous centres.

Prognosis.—This is very generally unfavorable. In only one case have I had reason to expect a cure. It often happens that amendment very decided in its character takes place soon after the beginning of the treatment with galvanism and barium. This has been the case in every instance of the disease that has been under my charge; but in only one has it been permanent. In those now under treatment, there has as yet been no relapse; but the time is too short to speak with any confidence in regard to the ultimate result.

Morbid Anatomy and Pathology.—The remarks made under this head, when the cerebral and spinal forms of sclerosis were being considered, apply to the cerebro-spinal variety. Charcot[1] has considered the subject of sclerosis mainly in its histological relations. The main points are—and these have already been stated several times—that the morbid process essentially consists in hypertrophy of the neuroglia at the expense of the proper nerve-substance, and that this is a consequence of inflammatory action. In the present form of the disease, the sclerosed tissue appears in the form of plates or nodules in different parts of the brain and spinal cord.

Treatment.—The treatment of multiple cerebro-spinal sclerosis is more palliative than curative. Galvanism to the brain and vertebral column, chloride of barium, and hyoscyamus, have very generally caused improvement for a time, but my experience goes to show that this is not permanent.

The galvanic current should be used of less tension when applied to the head, but as strong as the patient can endure, to the spine.

---

[1] *Gazette des Hôpitaux*, Nos. 102, 103, 140, 141, 143, 1868.

The chloride of barium should be given in solution in water, in doses of a grain three times a day—the hyoscyamus in doses of from one to two teaspoonfuls of the tincture.

I have sometimes given the nitrate of silver in fourth-of-a-grain doses, three times a day, and very generally recommend cod-liver oil with each meal. Occasionally I have administered iodide of potassium and the bichloride of mercury, with the view of counteracting a possible syphilitic diathesis.

Whatever measures are adopted should be continued for several months at least, and, if the improvement persists, for a much longer period.

CHAPTER VIII.

PARALYSIS AGITANS.

UNDER the term paralysis agitans, several affections have been included which are very different in character. I have already considered two of them—multiple cerebral sclerosis and multiple cerebro-spinal sclerosis; a third I propose to treat of under the name of paralysis agitans. Though the objections to its use are many, it possesses the advantages of being already known, and of expressing two of the main features of the disease to which it is applied. It is to be understood, however, that the disease to which I apply the term is altogether different from that which Charcot,[1] Ordenstein,[2] and other writers, call paralysis agitans.

The affection which Parkinson[3] described, and to which he applied the name "shaking palsy," has since been very carefully studied by many writers, and the fact has been clearly made out that it is not a single disease.

Charcot, in numerous memoirs and lectures to which reference has already been made, has very definitely shown that the affection which he designates *sclérose en plaques disséminées*—considered in this treatise under the name of multiple cerebro-spinal sclerosis—must be regarded as a distinct morbid condition; and in the first section of this work I have made the same claim for multiple cerebral sclerosis. The term paralysis agitans I apply to a very different affection from either, but one which I am confident will be recognized as presenting well-defined characteristics. Ordenstein[4] has included it with multiple cerebral sclerosis, and denies it any fixed seat; but Jaccoud[5] locates it in the pons Varolii, without, however, in my opinion, having any good reason for so doing. Of all writers Dr. Handfield Jones[1] appears to have the clearest ideas of the affection now under notice. Thus, he says:

---

[1] "Leçons sur les maladies du système nerveux," Paris, 1872-'73, p. 139.

[2] "Sur la paralysie agitante," etc., Paris, 1868.

[3] "Essay on the Shaking Palsy," London, 1817.

[4] *Op. cit.*

[5] *Op. cit.*, p. 424.

"It appears to me a question whether two distinct affections are not often comprehended under this name. For on the one hand it appears pretty certain that there is one form which is met with in old persons, is quite incurable, and is associated with, if not dependent on, organic wasting changes in the nervous centres; while another form occurs in younger persons, is more curable, and therefore is presumably not dependent on organic change." It is this latter disease which I propose to consider at present. The other embraces cases of multiple cerebral sclerosis and cerebro-spinal sclerosis.

Symptoms.—The primary manifestation is tremor, and this, like the same symptom in the severer forms of disease already considered, in which it forms an essential feature, may begin in a very restricted or more extensive region of the body. It is present whether voluntary movements are performed or not with the affected limbs, but is increased by mental excitement of any kind, by physical exertion, or by any cause capable of depressing the powers of the system.

It is not generally the case that the tremor shows any tendency to advance much beyond its original limits, however small or extensive these may be. When it does exhibit such a disposition, contiguous muscles are first attacked, and then the corresponding ones on the opposite side of the body.

From the very first there is muscular weakness, not to any very great extent, but still sufficiently evident to careful examination with the dynamometer. As the tremor increases in violence or extent, the paralysis becomes more obvious.

Sensibility is rarely affected, there is no bending of the body forward, no festination, and no head-symptoms. The tremor always ceases during sleep, except in very extreme and long-continued cases, and there may be intermissions of longer or shorter duration while the patient is awake.

Causes.—Paralysis agitans may result from emotional disturbance, from continuous or severe muscular exertion, from some exhausting disease, such as dysentery, typhoid or typhus fever, or rheumatism, or from blows, falls, or other injuries. In many cases the cause cannot be ascertained.

Of twenty-five cases of which I have records, ten were apparently due to mental causes, four to excessive physical exertion, four to diseases of various kinds, two to injuries, and in five no cause could be discovered.

Two cases of mercurial trembling, the symptoms of which affection are very similar to those of non-toxic paralysis agitans, are not included among the foregoing.

---

[1] "Studies on Functional Nervous Disorders," London, 1870, p. 382.

Diagnosis.—From multiple cerebral sclerosis, paralysis agitans is distinguished by the facts that there are no head-symptoms, no festination, and no derangements of sensibility. It is more apt to occur in persons under the age of fifty, and may be met with in quite young persons. The reverse of both these circumstances is true of multiple cerebral sclerosis.

From multiple cerebro-spinal sclerosis, it is diagnosticated mainly by the absence of any head-symptoms, by the fact that the tremor usually comes on before the paralysis, and is independent of voluntary movements.

From convulsive tremor it is readily distinguished by the facts that the tremor is not paroxysmal, and that it is accompanied by paresis of the affected muscles.

The character of the muscular action, and the history of the case, will prevent its being confounded with chorea.

Prognosis.—Paralysis agitans rarely terminates fatally, and when it does it is because the tremor has become so general that death results from exhaustion. It, however, often happens that all measures fail to relieve the agitation. Of the twenty-five cases occurring in my own experience, eight were cured, five partially so, and in the rest no permanent effect was produced by any means I employed.

Morbid Anatomy and Pathology.—Nothing is known of the morbid anatomy. In a few cases, patients have died either from the disease or from some intercurrent affection, and post-mortem examinations have been made with negative results. Petræus, quoted by Dr. Handfield Jones, relates two severe cases, one of which proved fatal. At the autopsy nothing was found but fatty degeneration of the heart and pneumonic consolidation of the right lung. He remarks on the tremor not being constant in many cases, ceasing for some days and then returning with fresh force, or changing its seat from one part to another.

In my opinion, the disease under consideration is due to an irregular and diminished evolution of nerve-force from the motor nerve-cells in relation with the nerves supplying the muscles in which the agitation exists. The pathology of tremor, not the result of structural lesions, is a subject which is beginning to be studied, but which is not yet clearly understood. We know that, when we have strongly exerted an arm, for instance, the muscles are tremulous for some time afterward, and that the agitation is rendered very evident when we attempt to write or do any other act requiring delicate muscular adaptation. A period of rest must take place before steadiness is regained. Now, in such a case the agitation is not probably due to any cause inherent in the muscle, but is the result of exhaustion in the nerve-cells and the disengagement of insufficient force in an intermittent manner. I suppose paralysis agitans to be due to some such action in the motor nerve-cells in the gray matter of the spinal cord.

In those cases in which the tremor becomes permanent, structural lesions of profound character—as in permanent hysterical contractions and epilepsy—doubtless occur.

Treatment.—I have used electricity, both of the galvanic and faradaic kinds, in all the cases of paralysis agitans that have been under my charge, and in conjunction have employed many internal medicines, such as arsenic, iron, manganese, zinc, copper, phosphorus, strychnia, and sedatives of various kinds, including opium, bromide of potassium, conium, stramonium, Indian hemp, and many others. I am very decidedly of the opinion that the best treatment consists in the use of the constant primary current to the spinal cord, sympathetic nerve, and the affected muscles, while at the same time strychnia and phosphorus, according to the formula given on page 58, are administered internally. By these means four of my eight successful cases were entirely cured within two months. One of these was sent to me by my friend Dr. F. N. Otis. The affection was confined to the right arm, and was probably due to inordinate gymnastic exercise; the other was a gentleman from St. Louis, in whom the disease was also confined to the right arm, and had apparently resulted from writing excessively. Both had lasted several months.

Another was a railway engineer, in whom the disease was the result of over-mental excitement; and the fourth was a distinguished clergyman of the Catholic Church in whom a like origin existed.

The six other cases were, two of them, consequent on other diseases, and four were without known cause. Three were women; the tremor in two was in both arms, and in two in one leg in each. The duration of the treatment was from three to seven months. A full and nutritious diet, and the avoidance of all mental excitement or strong physical exertion, are important features in the treatment.

CHAPTER IX.

ANAPEIRATIC PARALYSIS.

There is a class of paralyses produced by the habitual use of a particular class of muscles in the same way for a long time. Thus we have writer's paralysis, telegrapher's paralysis, hammer paralysis, and so on. To describe these as separate and distinct affections is scarcely, in the present state of our knowledge, permissible. I shall, therefore, embrace these under the designation of anapeiratic (Αναπειράω, *to do or* are very similar to those of non-toxic paralysis agitans, are not included among the foregoing.

Diagnosis.—From multiple cerebral sclerosis, paralysis agitans is distinguished by the facts that there are no head-symptoms, no festination, and no derangements of sensibility. It is more apt to occur in persons under the age of fifty, and may be met with in quite young persons. The reverse of both these circumstances is true of multiple cerebral sclerosis.

From multiple cerebro-spinal sclerosis, it is diagnosticated mainly by the absence of any head-symptoms, by the fact that the tremor usually comes on before the paralysis, and is independent of voluntary movements.

From convulsive tremor it is readily distinguished by the facts that the tremor is not paroxysmal, and that it is accompanied by paresis of the affected muscles.

The character of the muscular action, and the history of the case, will prevent its being confounded with chorea.

Prognosis.—Paralysis agitans rarely terminates fatally, and when it does it is because the tremor has become so general that death results from exhaustion. It, however, often happens that all measures fail to relieve the agitation. Of the twenty-five cases occurring in my own experience, eight were cured, five partially so, and in the rest no permanent effect was produced by any means I employed.

Morbid Anatomy and Pathology.—Nothing is known of the morbid anatomy. In a few cases, patients have died either from the disease or from some intercurrent affection, and post-mortem examinations have been made with negative results. Petræus, quoted by Dr. Handfield Jones, relates two severe cases, one of which proved fatal. At the autopsy nothing was found but fatty degeneration of the heart and pneumonic consolidation of the right lung. He remarks on the tremor not being constant in many cases, ceasing for some days and then returning with fresh force, or changing its seat from one part to another.

In my opinion, the disease under consideration is due to an irregular and diminished evolution of nerve-force from the motor nerve-cells in relation with the nerves supplying the muscles in which the agitation exists. The pathology of tremor, not the result of structural lesions, is a subject which is beginning to be studied, but which is not yet clearly understood. We know that, when we have strongly exerted an arm, for instance, the muscles are tremulous for some time afterward, and that the agitation is rendered very evident when we attempt to write or do any other act requiring delicate muscular adaptation. A period of rest must take place before steadiness is regained. Now, in such a case the agitation is not probably due to any cause inherent in the muscle, but is the result of exhaustion in the nerve-cells and the disengagement of insufficient force in an intermittent manner. I suppose paralysis agitans to be due to some such action in the motor nerve-cells in the gray matter of the spinal cord.

In those cases in which the tremor becomes permanent, structural lesions of profound character—as in permanent hysterical contractions and epilepsy—doubtless occur.

Treatment.—I have used electricity, both of the galvanic and faradaic kinds, in all the cases of paralysis agitans that have been under my charge, and in conjunction have employed many internal medicines, such as arsenic, iron, manganese, zinc, copper, phosphorus, strychnia, and sedatives of various kinds, including opium, bromide of potassium, conium, stramonium, Indian hemp, and many others. I am very decidedly of the opinion that the best treatment consists in the use of the constant primary current to the spinal cord, sympathetic nerve, and the affected muscles, while at the same time strychnia and phosphorus, according to the formula given on page 58, are administered internally. By these means four of my eight successful cases were entirely cured within two months. One of these was sent to me by my friend Dr. F. N. Otis. The affection was confined to the right arm, and was probably due to inordinate gymnastic exercise; the other was a gentleman from St. Louis, in whom the disease was also confined to the right arm, and had apparently resulted from writing excessively. Both had lasted several months.

Another was a railway engineer, in whom the disease was the result of over-mental excitement; and the fourth was a distinguished clergyman of the Catholic Church in whom a like origin existed.

The six other cases were, two of them, consequent on other diseases, and four were without known cause. Three were women; the tremor in two was in both arms, and in two in one leg in each. The duration of the treatment was from three to seven months. A full and nutritious diet, and the avoidance of all mental excitement or strong physical exertion, are important features in the treatment.

CHAPTER IX.

ANAPEIRATIC PARALYSIS.

There is a class of paralyses produced by the habitual use of a particular class of muscles in the same way for a long time. Thus we have writer's paralysis, telegrapher's paralysis, hammer paralysis, and so on. To describe these as separate and distinct affections is scarcely, in the present state of our knowledge, permissible. I shall, therefore, embrace these under the designation of anapeiratic (Αναπειράω, *to do or* *attempt again*) paralysis, as being caused by the frequent repetition of some particular muscular action.

Symptoms.—The first symptom usually observed is a feeling of fatigue experienced in the muscles which have been grouped together for frequent use in some especial way. Thus in writers, engravers, violinists, type-setters, and telegraphers, the tired sensation is felt in the muscles of the hand, forearm, arm, and shoulder. The thumb is especially affected, and is also the seat of a dull, aching pain. Pains, not very severe or fixed, are also common in the muscles higher up; this fatigue the patient endeavors to correct by grasping the pen or burin, for instance, more firmly, or by making an intense mental effort to regulate the muscular contractions by which the instruments are held, the type seized, or by which the fingers are moved over the strings of the violin, or the lever of the telegraph-instrument. But he only thereby adds to the difficulty, for the weariness and pain are increased, the muscles become weakened, and moreover irregular and incoördinate actions ensue which render the results of either writing, engraving, etc., more or less imperfect.

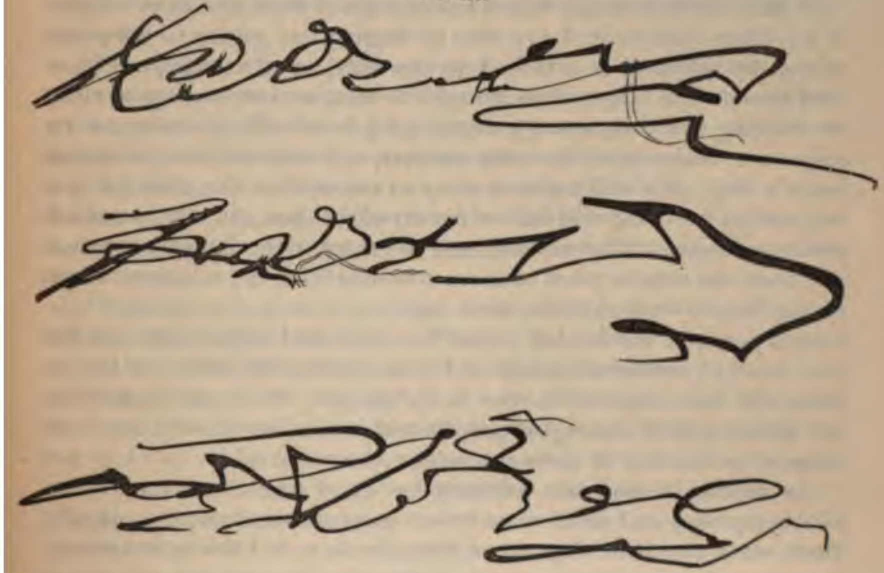
If he perseveres day after day in his occupation he soon reaches that stage of the disease in which the ability to direct the pen, for instance, in accordance with his will, is lost, and the automatic actions, which are of great importance in writing, are likewise very much diminished. For a time, then, he writes better when his mind is not occupied in directing the formation of every letter, but in which he allows the muscles as it were to take care of themselves. Constantly, however, he feels the necessity of mental action, and this action invariably increases the trouble, until, at last, the moment the attempt is made to write, the pen, actuated by the muscles of the fingers, executes such disorderly movements as to bear, in extreme cases, little or no analogy to the words attempted to be written. A distinct paroxysm is thus induced, which lasts as long as the patient persists in the attempt to write. When he discontinues, the spasm ceases, and he can perform any other act with the fingers without there being the slightest convulsive movements. In some cases there is pain in the fingers, the muscles between the metacarpal bones, and in those of the forearm. The spasm is much worse if the patient be excited or particularly anxious to do his best.

In the accompanying woodcut (Fig. 110) are represented three attempts of a patient to write the name "James Ely." At first some resemblance to the letter *J* is made, but in the second trial it is less distinct, and in the third is lost altogether.

All of my patients had resorted to various expedients to obviate the spasms, under the idea that they were produced by metallic pens carrying off the electricity from the arm; several had, for a time, made use of quills, or hard rubber pens, and for a time relief had been obtained, but the paroxysms soon became as bad as ever. Others had used very thick pen-holders, and this expedient was also, for a time, successful. In the end, however, all such efforts to prevent the spasms proved futile.

In one case under my charge, the patient, an engraver, was utterly incapable of using his burin, although he could write for hours perfectly well, and those who had contracted the disease by excessive writing could execute any other delicate movements, such as drawing, playing the piano or violin, threading needles, etc., without inconvenience. In several cases the individuals had acquired the power to write with the left hand, but before long this was also affected.

FIG. 110.



Dr. G. V. Poore[1] has recently published an interesting memoir on the affection as produced by excessive writing, and argues that, although it is true that patients can execute other actions than writing with the affected hand, the muscles employed in these movements are not the same as those used in writing. This is doubtless true of advanced stages of the disease, but it certainly is not so of early periods. I have a patient at this time under my charge who cannot write without great inconvenience, but who uses a pencil in drawing with the greatest facility and precision.

Dr. Frank Smith[2] describes the disease I have designated anapeiratic paralysis, as it occurs in workmen who use the hammer almost continually in certain processes, and gives it the name of hephæstic (Ηφαιστος, *Vulcan*) hemiplegia.

---

[1] "Writers' Cramp, its Pathology and Treatment," *The Practitioner*, June, July, and August, 1873.

[2] *Lancet*, March 27, 1869, also "On Hephæstic Hemiplegia or Hammer Palsy," *British Medical Journal*, October 31, 1874.

"There are numerous varieties of manufactures in which the rapid use of a light or heavy hammer plays a chief part, such for example as table-blade forging, scissors-making, saw-straightening, razor-blade striking, engineering, file-forging, etc."

"The pen-blade forger uses a hammer about three pounds in weight. A pen-blade receives in the process of forging and joining to the piece of iron by which it is attached to the haft, on an average, one hundred blows. The forger, if an industrious man, anxious perhaps to save, by working overtime, enough money to join a building-society, or to commence business on his own account, will work twelve or thirteen hours a day. He will make as many as twenty-four dozen blades in a day, and in so doing will deliver twenty-eight thousand eight hundred accurate strokes. The rapidity and accuracy with which these blows rain upon the slender piece of iron are wonderful to the onlooker. Supposing him to work three hundred days in a year, and to continue this for ten years, he will in that period have delivered eighty-eight million four hundred thousand strokes, and just so many discharges of nerve-force will have occurred in the motor ganglia which are engaged in the action, and in the higher ganglia which calculate the distance and judge of the amount of force necessary to be evolved."

In several of the cases adduced by Dr. Frank Smith there were head-symptoms, and in all more or less extensive hemiplegic paralysis. There were also twitchings of muscles, pains, and difficulty of speech, in some of the cases.

M. Onimus[1] was, I think, the first to call attention to the disease as met with in telegraph-operators. The trouble appears usually to manifest itself in the first place by a difficulty in coördinating the muscles so as to make dots or points with the instrument. After a time the same restraint is experienced in the formation of lines. The disease appears to be rare in this country, which—as, according to M. Onimus, the Morse machine is especially apt to induce it—is somewhat remarkable.

In several of my cases there have been symptoms indicative of disorder of the central nervous system. These have consisted of headache, pain in the back, and occasional tremors of the limbs. In one case there is marked inability to coördinate the muscles of articulation so as to speak clearly. The trouble seems to be more in the lips than in the tongue, and there is decided mental impairment. In this case there is no doubt that the affection has originated from the excessive uses of the muscles of the right hand and arm in writing.

Causes.—The disease is more apt to attack persons somewhat advanced in life, than the very young. All my patients were over forty years of age. All were males, though this proclivity of men to the affection is not absolute, as several cases are on record in which women, seamstresses especially, have been its subjects. It is apparently sometimes induced by using the fingers in constrained positions. In one of my cases, the patient, who had been in the habit of writing with the hand supported by the little finger, cured himself by allowing the whole hand to rest on the desk. The principal cause—the habitual performance of certain restricted movements—has already been sufficiently considered.

---

[1] *Gazette Médicale;* also *Chicago Journal of Nervous and Mental Diseases*, 1875.

The opinion which Poore expresses, that it is due in writers to the use of steel-pens, is not borne out by my experience. I have seen it in persons who always wrote with quill-pens, and, as we know, the disease occurs in individuals from other causes than writing.

Diagnosis.—Attention paid to the characteristic symptoms of anapeiratic paralysis, and inquiry into the clinical history, will prevent its being mistaken for lead-paralysis, progressive muscular atrophy, or any other disease.

Prognosis.—In the early stage anapeiratic paralysis, by whatever cause induced, admits of cure. When it has existed a long time, and when the patient cannot rest, a cure is almost impossible.

A majority of the cases that have come under my notice had lasted too long to admit of cure, and the patients had, notwithstanding the imperfections of their work, persisted in using the affected muscles in the actions which had led to the causation of the disease, and then when this was no longer possible had used the other hand in like manner, till it also had become affected. In such cases permanent cures are almost out of the question, although relief can be obtained to such an extent as to allow of occasional writing.

Morbid Anatomy and Pathology.—As regards the morbid anatomy, there are no data, and the lesion is probably not one which can be detected by our present means of observation. The affection is, however, doubtless due to disorder in the normal action of the motor cells, and this disorder is the result of over-exertion of a particular set of muscles in a particular way. Examples of cerebral exhaustion by the predominance of one idea, or a series of ideas for a long time, are often witnessed. Writer's spasm is, I conceive, the result of a similar action in spinal motor cells and cerebral nerve motor centres.

Poore, however, does not believe that the affection, as met with in writers, can be of central origin, but certainly the symptoms are of a character to militate against his view. He has looked at the disease from too restricted a stand-point. No one can read the report of Dr. Frank Smith's cases without at once perceiving that they are the results of central lesions.

Treatment.—The most indispensable means of cure is rest, and, unless this can be secured, it is useless for the physician to undertake the treatment. In some cases it has succeeded without any assistance. The abstinence from the labor causing the disease, and sometimes from all continuous muscular exertion, should be absolute during at least six months.

The constant galvanic current has proved the most effectual agent in my hands: I apply it to the sympathetic nerve, the spinal cord in its upper part, and to all the muscles and nerves of the upper extremity. A half an hour three times a week, with a current of considerable intensity (forty cells), will be sufficient. Faradization, in my experience, is more productive of harm than benefit.

With the galvanism I have administered the combination of phosphide of zinc, and extract of nux-vomica, recommended on page 54 of this treatise.

The bromide of zinc in incipient cases is a most efficacious agent in restoring tone to the nervous system, and in conjunction with rest will often effect a cure. It should be used in gradually-increasing doses as recommended for convulsive tremor and chorea.

When a cure cannot be effected, well-devised prothetic apparatus will enable the patient to write or perform other actions requiring skill rather than strength; but I am not sure that they do not lead to the further extension of the disease, especially in its cerebral relations. Division of tendons or muscles is not admissible.

CHAPTER X.

EXOPHTHALMIC GOITRE.

It is with hesitation that I have ventured to include the remarkable disorder called Graves's disease, Basedow's disease, exophthalmic goitre, and by several other designations, under the head of cerebro-spinal affections. But, after a careful consideration of all the points in its clinical history and morbid anatomy, as they have been observed by others, and studied by myself, I find it difficult to place it in any other category. The reasons which have governed me in this decision will be stated under another division of this chapter.

Symptoms.—The first phenomenon to make its appearance, in a case of exophthalmic goitre, is irregular and excessive action of the heart. The organ is far more irritable than when in a state of health, and thus slight emotional disturbance or moderate physical exertion readily affects its action. Even when the patient is mentally and physically quiet, the pulsations are rarely below a hundred in a minute, and the least excitement, mental or bodily, will send them up to a hundred and twenty, a hundred and fifty, a hundred and sixty, or more, in extreme cases.

With the increase in frequency there is generally an augmentation of the force of the heart. The patient feels its pulsations against the wall of the chest, feels them as the whole body is shaken by them, and hears them in the murmur which is constantly in the ears.

The carotids and abdominal aorta can be seen to have their action increased, and the jugular veins, always dilated, are sometimes the seat of pulsation.

Physical examination does not in general indicate the existence of any organic disorder. Sometimes, however, the heart is found to be enlarged, but rather as a consequence than a cause of the disturbance.

A systolic murmur is often heard, which may be either arterial or ventricular. In the former instance it is anæmic, in the latter it is due to a relative insufficiency of the auriculo-ventricular valves.

The next symptom in order is usually an enlargement of the thyroid gland, an enlargement which is variable, and which is greater or less in accordance with the excessive or moderate action of the heart. Notwithstanding this capacity for change in size, there is a permanent augmentation in the volume of the body, below which the decrease does not take place.

If the hand be laid over the swollen thyroid, a peculiar sensation like that derived from stroking a purring cat—*frémissement cataire*—is felt with every systole of the heart, and a bellows-murmur is heard when the ear is applied to the part.

Next, the third essential phenomenon makes its appearance, and this consists of a prominence of the eyeballs. Usually this is symmetrical, but occasionally one protrudes more than the other. In the early stage of the affection the lids can be closed over the eyes, but in extreme cases they cannot be brought together, and the conjunctivæ are therefore exposed to the atmosphere and to particles of dust, which cause excessive lachrymation and sometimes troublesome inflammation.

The pupils rarely exhibit any deviation from the healthy state. I have sometimes found them abnormally dilated, never contracted, and always sensitive to light.

Graefe[1] has called attention to a circumstance which accompanies the protrusion of the eyeballs, and that is the disassociation of the movements of the upper eyelid from those of the eyes. In the normal condition, when the globe of the eye is raised, the lid is also elevated, and when the globe is depressed the lid likewise falls. In exophthalmic goitre these automatic movements do not take place.

These three phenomena, excessive action of the heart, enlargement of the thyroid gland, and protrusion of the eyeballs, may be said to constitute the pathological trinity of which the disease consists, but there are cases in which the goitre is scarcely if at all present, and others in which the exophthalmos is absent, and probably others, again, in which both these phenomena are wanting.

---

[1] "Bemerkungen über Exophthalmos mit Struma und Herzleiden," *Archiv für Ophthalmologie*, 1857.

Again, there is no definite relation between the degrees of severity characterizing these symptoms. Sometimes the heart is most tumultuous in its action, the goitre large, and the eyes very slightly prominent, or the eyeballs may protrude to the utmost and the goitre be small, and the heart not excessively deranged, and so on.

But though these three phenomena constitute the most marked features of the disease, there are others which, though not so obvious to others, add greatly to the distress of the patient. Thus there are generally cough, nausea, œdema of the extremities, increase of temperature, profuse sweating, and occasional hæmorrhages from the nose, lungs, or bowels.

The emotional excitability I have always found increased, sleep is generally disturbed and insufficient, there are headache, vertigo, and noises in the ears, the character often undergoes a marked change, and individuals who were quiet and gentle become excited, suspicious, and irritable.

Quite recently Dr. Bulkley,[1] of this city, has reported two cases in which there was urticaria.

Anæmia is generally the predominant physical condition, and with it there is more or less mental weakness. The body is usually much emaciated, probably in part from defective appetite and defective assimilative power, which ordinarily exist. In woman, the menstrual discharge is almost always either entirely suppressed or greatly diminished.

It rarely happens that there are any marked disturbances of vision, and the movements of the eyeball do not appear to be impeded. The fundus of the eye, when examined with the ophthalmoscope, is generally found to be normal; occasionally there are venous dilatation and pulsation.

For the following history of a case of exophthalmic goitre, and the accompanying illustration from a photograph, I am indebted to Dr. J. B. Crawford, of Wilkesbarre, Pa. The case is particularly interesting from the fact that it occurred in a man, was remarkably acute in character, terminated fatally, and that, notwithstanding the excessive action of the heart during life, there was no cardiac hypertrophy.

"*July* 2, 1872.—Visited Colonel E. B. H., occupation, lawyer, fifty-three years of age, and of nervo-sanguine temperament. Has been afflicted with muscular rheumatism for ten years, contracted in military service in Virginia, in 1861 and 1862. Has been engaged in active business until within the past two weeks. He has at times been conscious of rapid and forcible beating of the heart, increased by either physical or mental effort. During the past two months this has been steadily increasing in severity. He has had much pain in the abdomen for a long time. Has had a slight cough and expectoration for more than a year.

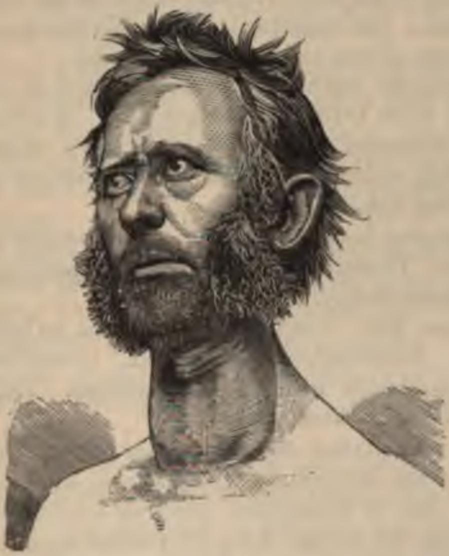
---

[1] "Two Cases of Exophthalmic Goitre associated with Chronic Urticaria," *Chicago Journal of Nervous and Mental Disease*, October, 1875, p. 513.

"About six weeks ago he first observed a distinct enlargement of the thyroid gland. He remembered, however, that during the past year or more, he has had difficulty in buttoning his shirt-collar.

"The gland is now two and a half inches in diameter, and very prominent. He has marked prominence of the eyes, giving to his features a staring, wild expression. (Fig. 111.) The eyeballs seem projected directly forward. There is no strabismus, nor perversion of sight. The eyelids are scarcely sufficient to cover the eyeballs. Slight compression returns the eyes to their normal position in their sockets; but upon removal of pressure they are immediately protruded to the extent of their former prominence. The lachrymal secretion is as free as usual. The action of the heart is exceedingly violent, its beating being distinctly observable by the movements of the patient's clothing, and numbering 123 per minute.

Fig. 111.



"Examination by percussion and with the stethoscope discloses no symptoms of hypertrophy nor evidence of valvular lesion. Area of precordial dullness not increased. Distinct bellows-murmur is heard over the left ventricle—much more distinct over the arch of the aorta and left carotid. Fine venous murmur is heard over both thyroids, and distinct arterial impulse observed over abdominal aorta on palpation.

Breathing is vesicular, respiration twenty per minute. The skin is pale, the face becoming flushed when under mental excitement. The lips and membrane lining the mouth exceedingly pallid. The bowels are constipated. The patient has slept very little during the past four months. He appears nervous and agitated. His hands are exceedingly tremulous. He has lately found it difficult to write legibly or to even hold a pen.

"A saline cathartic was prescribed—one-sixtieth grain of aconitia, to be given every six hours. Diet to be light and nutritious. Quiet, mental and physical, was enjoined.

"*3d.*—Patient slept several hours last night. Bowels have acted freely. He feels better. Pulse, 120 per minute.

"*5th.*—Has rested well. Pulse, 100. Treatment continued.

"*7th.*—Pulse, 90 per minute. Has had short paroxysms of palpitation, but no pain. His appetite is poor. Treatment continued, with addition of citrate of iron and extract of gentian.

"*9th.*—Symptoms unchanged. Examination of urine shows its specific gravity to be 1019, and strongly acid. A few small tube-casts are noticed. Numerous small crystals of triple phosphate, quantity normal. Aconitia continued. Elixir pyro-phosphate of iron and cinchona, one drachm before each meal.

"*12th.*—Has slept better. Pulse, 95. No recurrence of palpitation. Appetite better. Protrusion of eyes less conspicuous. Patient rode out a short distance to-day.

"*15th.*—Pulse, 100. Condition nearly same as before. Temperature, 98° Fahr. Exophthalmia less marked. Gradual emaciation.

"*17th.*—Consultation with Dr. C. Washburn. Patient's condition unchanged. Treatment continued.

"*20th.*—In consultation with Drs. Washburn and Rothrock, it was decided to give the following: ℞. Spir. æth. co., ℥iv; hydrocyanic acid, ʒj. M. Sig. A teaspoonful every six hours. About midnight, soon after taking the second dose, the patient became delirious. His son, Dr. O. F. Harvey, who was with him at the time, states that the patient's face became flushed, his head hot, his feet and hands cold. The pulse rose to 115 per minute.

"Ice-water was applied to his head, and hot applications to his extremities. Delirium continued about one hour. I was sent for, and ordered the last-prescribed medicine to be discontinued. Aconitia to be given as before, with elixir valerianate of ammonia, one drachm, every three hours.

"*21st.*—Patient able to sit up and walk about the room. Pulse, 95. Appetite improved.

"*22d.*—Symptoms improved. Pulse, 90.

"*31st.*—Patient growing weaker; otherwise but little changed. My own ill-health making it necessary for me to leave town for a while, the patient is left in care of Dr. Washburn until my return. The following memoranda were made by him:

"*August* 1*st.*—Patient about the same as yesterday. Resting very poorly.

"3*d.*—Changed treatment from aconite to digitalis.

"5*th.*—Not doing well under treatment with digitalis. Changed back again to aconite. Patient is directed to take a tablespoonful of whiskey in a wineglassful of milk whenever desired.

"7*th.*—The whiskey has made patient feel more comfortable.

"9*th.*—No marked change. Patient seems to be at a stand-still.

"11*th.*—Patient very much emaciated and weaker; is scarcely able to expectorate the considerable mucous secretion which accumulates about the trachea and throat.

"13*th.*—Milk and whiskey are the only food which the patient's stomach will retain; to be given freely.

"15*th.*—Returned home to-day, and again took charge of patient. Emaciation has increased. He is much weaker than when I last saw him, otherwise but little changed. He takes no food except a little milk and whiskey, and sleeps very little, and that, he says, gives him no rest, and does not refresh him. The pulse is variable, ranging from 90 to 100 per minute.

"16*th.*—Has had an attack of severe pain in the left side of chest. Chloroform-liniment to be applied. One-sixth grain of sulphate of morphia to be given every three hours until relieved.

"17*th.*—Patient feels easier. Debility increased.

"20*th.* 10 A. M.—Patient has great difficulty of breathing in the recumbent position. Extremities cold. Pulse imperceptible at the wrist. Died at 1.30 P. M.

"22*d.* *Post mortem.*—Rigor mortis well marked. The eyeballs and thyroid gland exhibit but little of their former prominence. Lungs healthy, except some old adhesions on left side; a moderate amount of effusion in left pleura; liver healthy; gall-bladder very small; stomach, spleen, and intestines healthy; heart healthy in appearance, *somewhat below the average size, valves perfect;* aorta greatly enlarged through its whole extent, its calibre being one and a half inch in diameter; arteries generally enlarged (or dilated); kidneys moderately hypertrophied, and much congested; thyroid gland much enlarged and apparently normal in texture. General emaciation extreme. Nervous system not examined."

Causes.—Exophthalmic goitre is far more frequent in women than in men. Of the eleven cases occurring in my experience, all were in women. Eulenburg[1] states the relation of women to men as two to one. Romberg and Hennock,[1] of twenty-seven cases, found twenty-four in females, and Cheadle,[2] but one male in nine cases.

---

[1] "Die Basedow'sche Krankheit," Ziemssen's "Handbuch," u. s. w., zwölfter Band, II., zweite Hälfte, Leipzig, 1875, p. 75.

The disease is more frequent between the ages of twenty and forty than at any other period. All my cases were within these limits. Instances, however, have been reported occurring both in younger and older persons. Men are stated by Jaccoud to be more frequently affected after thirty years of age, and women under this age.

Among the exciting causes mental shock is probably the most efficient. Four of my cases originated from this influence. It has been known to be developed almost instantaneously by powerful emotional disturbance.

Dr. Begbie [3] has reported a case in which the disease was apparently caused by a wound of the head, and Graefe has adduced a like example.

Diagnosis.—There is not much danger that exophthalmic goitre will be mistaken for any other affection by a physician familiar with its very pronounced characteristics. The excessive action of the heart, the enlargement of the thyroid gland, the prominence of the eyeballs, the anæmic condition of the system, the venous murmurs, all go to make up a pathological picture, of which the elements are sufficiently well marked. But there are sometimes cases of the disease met with, in which some of the phenomena are not very decidedly shown, and, in these, care should be exercised before a definite opinion is pronounced. The facts that there are not the physical signs of organic lesion of the heart, notwithstanding its excessive action, that the swelling of the thyroid communicates a peculiar thrill to the hand, and that the protrusion of the eyeballs is on both sides and is unaccompanied with disturbance of vision, will even in doubtful cases serve to render the diagnosis sure.

Prognosis.—It is not often the case that exophthalmic goitre proves fatal. A few such cases are, however, on record, and therefore the prognosis, even as regards life and death, should be somewhat guarded. Relative to a cure being effected, there is still more uncertainty, though I am inclined to think that with proper treatment exophthalmic goitre is not a very intractable affection; the prospect of a mitigation of the severity of the phenomena may be reasonably held out in almost every case. Of the eleven cases occurring in my experience, four were permanently cured, and six more or less completely benefited. One only, a young lady from Astoria, and the most extreme instance of the disease I have witnessed, resisted all treatment.

Morbid Anatomy and Pathology.—Lesions have been found in the brain, the spinal cord, and the sympathetic nervous system, and in other cases there have been no appreciable alterations discovered in any one of these centres. The opinion seems to prevail that the seat of the disease is in the sympathetic nerve, especially the cervical portion, and several instances in which this part of the nervous system was the seat of morbid process have been reported by Traube and Recklinghausen,[1] Trousseau and Peter,[2] Archibald,[3] and others.

---

[1] Romberg, "Klinische Wahrnemungen und Beobachtungen," Berlin, 1851.

[2] "Exophthalmic Goitre," "St. George's Hospital Reports," vol. iv., 1869, p. 175.

[3] *Edinburgh Medical Journal*, 1849.

On the other hand, the brain and spinal cord have been found diseased in several cases. In one reported by Prael, cerebral embolism was the lesion, and in another by Geigel,[4] although the cervical sympathetic was the seat of alteration, the upper part of the spinal cord was in a state of sclerosis.

Again, Paul,[5] and Fournier and Ollivier,[6] have given the details of post-mortem examinations made in two cases of the disease in question, in which the sympathetic and all its ganglia were in a normal condition.

I am inclined to think that in the present state of our knowledge we are scarcely warranted in locating exophthalmic goitre in the sympathetic nervous system, and that we are justified in regarding it as an affection of the brain and medulla oblongata. I base this opinion not so much on the fact that there have been cases in which no lesion of the sympathetic has been found—though, notwithstanding our imperfect means of research, the point is of some importance—as on the nature of the symptoms which characterize the disease. The disturbance of cardiac action, the cough, nausea, and protrusion of the eyeballs, indicate the pneumogastric nerves as the organs through which the phenomena are manifested, and, though the enlargement of the thyroid gland shows vaso-motor disturbance, this can undoubtedly be produced from a lesion of the medulla oblongata as well as from one of the sympathetic. The absence of pupillary disturbance is one of the strongest circumstances against the hypothesis of sympathetic disorder.

Vulpian[7] has fully discussed the question of the anatomical substratum of exophthalmic goitre, and concludes that it is not yet shown that this is to be found in the sympathetic nervous system. The fact that we cannot produce a corresponding affection in the lower animals by irritation of the sympathetic nerve, is strongly against the correctness of the theory under notice.

Many have held the opinion that the disease is an affection primarily of the heart and the blood-forming organs, and they adduce the chloro-anæmia which so generally exists as a prominent condition, in support of this view. I was at one time disposed to accept this as the most reasonable explanation of the pathology of exophthalmic goitre, but more thorough consideration has served to convince me that it is not tenable. We have, in fact, no evidence to show that either chlorosis or anæmia ever produces in their entirety the remarkable phenomena characteristic of Graves's disease; on the contrary, we see every day the most well-marked examples of both affections without either thyroid hypertrophy or ocular prominence.

---

[1] "Deutsche Klinik," 1863.

[2] *Gazette Hebdomadaire*, 1864.

[3] *Medical Times and Gazette*, 1865.

[4] *Archiv für Ophthalmologie*, 1857.

[5] *Berliner klinische Wochenschrift*, 1865.

[6] *Gazette Hebdomadaire*, 1867, and *Union Médicale*, 1868.

[7] "Leçons sur l'appareil vaso-moteur," Paris, 1875, tome ii., p. 645

Neither can it be regarded as due to an organic affection of the heart. It is true that sometimes this organ is found to be the seat of structural disease in cases of exophthalmic goitre, but these lesions—generally hypertrophy and mitral insufficiency—are themselves rather the results than the cause, and moreover they are not such as could, so far as our knowledge extends, produce either enlargement of the thyroid gland or protrusion of the eyeballs.

As regards the morbid anatomy of the organs which are the seat of the more prominent symptoms of the disease, a few words are necessary in addition to those already given relative to the heart.

This enlargement of the thyroid is due not to any proliferation of its proper tissue, but entirely to the increased quantity of blood entering its vessels and distending it as water distends a sponge. The vessels, therefore, are always found enlarged far beyond their normal dimensions.

The prominence of the eyeballs has been supposed to result from vascular turgescence in the orbit, to an increase in the amount of fat which this cavity normally contains, and to a fatty degeneration of the ocular muscles by which their tone is destroyed, and the eyeball allowed to protrude. These causes probably contribute to the production of the phenomenon. We must, however, add another still more influential, and that is the contraction of Müller's orbito-ocular muscle by which action the eye is actively thrust forward.

Treatment.—The internal medication most to be relied on in my experience is that of which iron, some one of the bromides—zinc preferably—digitalis, and ergot, are the primary features. These may be combined as in the following formula: ℞. Ferri pyrophosphatis, zinci bromidi, āā ʒ j; digitalis tinct., ʒ v; ergotæ ext. fl., ℥ iv; M. ft. mist. Dose, a teaspoonful three times a day. In addition the patient should drink a glass or two of malt-liquor every day, and eat plentifully of animal food.

While I know of no better internal medication than this, I have occasionally, after keeping it up for several weeks, changed it for strychnia and phosphorus—the combination of extract of nux-vomica and phosphide of zinc, recommended on page 54—and with excellent results.

But this is by no means all or even perhaps the most important part of the treatment. The primary galvanic current is undoubtedly the principal agent to be relied on for the cure of the disease. I first used it successfully in 1865, in the cases of two sisters subject to exophthalmic goitre who were referred to me for treatment by Dr. J. W. Richards. The beneficial influence over all the symptoms was at once perceived, and in less than a month both were free from manifestations of the disease. Since then I have not treated a case without this remedy.

The intensity of the current should be as great as the patient can bear with comfort. This is the best rule ; any other based on the number of cells would be of uncertain import, owing to the exhaustion which takes place through use of the battery. One pole—a wet sponge—should be placed on the nape of the neck, and the other should be stroked down the skin over the pneumogastric—or sympathetic if the physician regards this nerve as the seat of the disease: it is impossible to act on the one without at the same time affecting the other to an equal extent. This should be done daily for five or ten minutes.

It is well, also, to pass the current through the hypertrophic thyroid.

Prof. Roberts Bartholow[1] has recently reported three cases in which the primary galvanic current was used with success. In these instances the negative pole was placed on the epigastrium, and the other so applied as to include the cervical sympathetic, the pneumogastric, and cilio-spinal region, within the circuit. Each *séance* was of about ten minutes' duration, and amendment was noticed from the first.

In six cases I have injected the fluid extract of ergot into the enlarged thyroid, in addition to giving it internally, and using galvanism. In all, the effect in reducing the size of the tumor was well marked. In the first two cases I injected only twenty minims, in one dose daily; but, in the others, I found by experience the advantage of making multiple injections, employing for each from twenty to thirty minims, and puncturing the tumor at various points.

Various other remedies have been proposed—iodine, opium, hydrotherapeutics, etc.—but, although there is no objection to their use, I am satisfied that they should not be employed to the neglect of the measures recommended.

---

[1] *Chicago Journal of Mental and Nervous Diseases*, July, 1875.

SECTION IV.

DISEASES OF THE PERIPHERAL NERVOUS SYSTEM.

I DO not propose to include under this head all the diseases to which the several nerves of the body are liable. Many of them are more appropriately considered in special treatises devoted to the eye and ear, and others differ merely in their situation, the essential condition being the same. Thus any nerve of the body may be paralyzed from injury, a disease, or from some contiguous affection capable of interfering with the due performance of its functions. It would scarcely be necessary in a general treatise like the present to give such paralyses separate consideration, as their features and the treatment proper can be sufficiently pointed out under the head of a typical representative.

Besides, many affections which are often regarded as being located in the peripheral nervous system are really central in situation. Among these are various maladies characterized by paralysis, spasm, hyperæsthesia, and anæsthesia, which have already been considered as central diseases.

I shall divide the affections of the peripheral nervous system into two groups; those which are characterized by recognizable anatomical changes in the nerves—Organic Diseases; and those in which such changes are not discoverable—Functional Diseases. The four immediately following chapters describe known pathological conditions, and therefore organic diseases; the others relate to affections, or rather symptoms, which are sometimes the results of structural changes in the nerves, and are again apparently entirely functional. In the present state of our knowledge it appears to be impossible to be more exact.

CHAPTER I.

NEURAL CONGESTION.

CONGESTION of nerves is, as Mitchell[1] states, scarcely recognizable by clinical observation. My experience is limited entirely to a study of the phenomena exhibited by the affection artificially produced, and in these investigations I have followed the line which Mitchell has so thoroughly pursued.

If, as he has pointed out, a nerve-trunk be subjected to the action of intense cold so as to be frozen, the period of congelation is immediately followed by one of congestion, the result of the paralysis of the vaso-motor nerves of the part.

Thus, if the sciatic nerve of a rabbit, for instance, be exposed, and, while a thin sheet of India-rubber protects it from direct contact, the vapor of ether or of rhigolene be thrown upon it from a vaporizer, the functions of the parts below—sensation and motion—are abolished, and remain so while the congelation lasts.

But as the temperature rises a new set of phenomena ensues. The nerve loses its whiteness, and becomes pinkish, or even red, and this gradually disappears—without, so far as can be perceived, the animal suffering any marked inconvenience. But, if the operation be repeated, or if the congelation be continued for a long time, the nerve becomes permanently discolored, and the animal is rendered lame. If the nerve be examined with a lens, such for instance as one of those furnished with Nachet's simple dissecting microscope, the vessels are seen to be enlarged and more numerous than in the normal condition, and minute extravasations coming from the over-distended vessels are seen between the fibres.

In man, though we cannot observe the anatomical changes, we are able to study, subjectively, with fullness and exactness, the symptoms which are due to neural congestion.

Mitchell,[2] in reference to this point, says: "I have repeatedly chilled or frozen the ulnar nerve in myself with ice or ice-and-salt. The first effect is to cause intense aching pain, which, although most severe in the little finger, the outside of the third finger, and the ulnar palm, is also felt in the whole hand, and especially on the back of the hand at the space between the metacarpal bones of the thumb and forefinger. The pain rather suddenly ceases at a certain stage of freezing, and for a moment the hand feels natural. Then the ulnar distribution in the hand begins to be numb, and this increases till all sensibility is lost—touch, pain, and the thermal sense disappearing in turn. Last of all, motility, which very rarely is slightly affected, lessens by degrees and is lost altogether. Soon after the part grows numb, the thermometer rises slowly, sense of heat is felt in the ulnar palm, and this region in my own case sweats excessively. At the same time the ulnar nerve at the elbow grows very excitable, and the least tap on the nerve causes slight pain in the third and fourth fingers, and sudden flexion of the first phalanges of all the fingers save the first, as well as adduction of the thumb.

---

[1] "Injuries of Nerves and their Consequences," Philadelphia, 1872, p. 56.

[2] *Op. cit.*, p. 59.

"The average rise of the thermometer in moderate chilling which does not annihilate sensation, and leaves motion but slightly impaired, is 2° Fahr. In more complete freezing it is in my case from 3° to 4° Fahr.

"The symptoms which follow the thaw are, as I believe, due chiefly to congestion. The nerve remains sore at the elbow and even some distance below and above it, while the brachial plexus may become tender (Waller), and, as the thawing occurs, the heart may be enfeebled and syncope threaten (Waller), or vertigo occur, as I have felt in my own case. The terminal distribution of the nerve suffers, after severe freezing, for hours or days; the soreness of surface, numbness, prickling, and partial loss of power may continue, together with a certain fullness which is felt, and which makes itself visible to the eye. Even after slight freezing there may remain for hours certain uncomfortable sensations, which scarcely admit of distinct description. In one instance these symptoms endured for eleven days, according to Waller, and in my own case they usually lasted from ten to fourteen days."

I have several times, with the view of studying the resulting phenomena, frozen my left ulnar nerve by throwing upon the skin over it, where most superficial, the vapor of ether. I have not, however, been able to add much to the account of the symptoms given by Mitchell. By means of Lombard's instrument I have observed the rise of temperature spoken of by Mitchell, but have, I think, ascertained that in the very beginning of the operation the temperature is slightly decreased, and that it is not till the freezing process is well advanced that the temperature rises.

Congestion is probably the condition present in many cases of pain in nerves, which usually pass for neuralgia. This is, I think, especially apt to be the case when with the pain there is either clonic spasm or paralysis of certain muscles supplied by the affected nerve, or both these phenomena. It is also doubtless the primary condition of neuritis.

CHAPTER II.

ACUTE NEURITIS.

Symptoms.—When the affected nerve is superficial it may be felt as a hard cord under the skin, pressure upon which causes an aggravation of the pain of which it is always the seat. The skin over it is generally red, showing, therefore, the course of the nerve.

If the nerve is a compound one, the parts to which it is distributed are the seat of symptoms resulting from the disturbance of physiological function. There is pain, and there is either spasm or paralysis, or both. The pain in the nerve-trunk, as well as that in the parts which it supplies, is increased at night, and there may be sympathetic pains in other and distant parts of the body. As the morbid process advances, the tactile sensibility in the parts of distribution become less, and after a time may be entirely abolished, but the perception of pain is not lost.

Reflex excitability and electric contractility are diminished from the first or almost from the first, and the muscles supplied by the nerve undergo atrophy unless the disease soon subsides. The temperature of the parts to which the nerve is distributed is increased 3° or 4° Fahr.

If the inflamed nerve is only sensory in function, as, for instance, the ophthalmic branch of the fifth, the manifestations are mainly as regards sensibility, although even here motility, as shown by the occurrence of clonic spasms in the face, is reflectively disturbed.

In cases of inflammation of motor nerves, spasm and paralysis are the chief symptoms, the latter being the permanent condition should the functions of the nerve not be restored.

The skin covering the parts supplied by the diseased nerve is often the seat of an erythematous or bullous affection.

In one of Mitchell's cases there was sudden œdema developed within three days, and a week later neural arthritis.

It is rarely the case that acute neuritis ends in complete resolution. Mitchell never observed a case of the kind. Jaccoud, however, speaks of it as terminating either by complete cure, that is to say by a cessation of the pain and return of the normal functions of the nerve, or by the supervention of permanent anæsthesia or paralysis, or both, according to the function of the affected nerve.

The paralysis not infrequently met with as a consequence of long-continued exposure to cold is probably the result of neuritis. Duchenne[1] so regards it. Several cases of the kind have come under my notice, and the majority have been in the radial, the ulnar, and the posterior circumflex nerves. The symptoms were similar to those just detailed, except that there was little or no pain. Indeed, in non-traumatic acute neuritis the presence of these pains is quite an exceptional circumstance, whereas, in the secondary form of the disease resulting from traumatism, pain is a prominent characteristic.

---

[1] "De l'éléctrisation localisée," Paris, 1872, p. 692.

Causes.—Acute neuritis is not often met with as an idiopathic affection. Generally, it is caused by wounds or injuries, or, as in a case mentioned by Mitchell, by the extension of cancerous ulceration. It appears, however, sometimes to be very difficult to excite even by extensive injuries, or by exposure to the action of the atmosphere or other extraneous agents. I have repeatedly seen the trunks of large nerves exposed and subjected to irritations of various kinds, both in man and in the lower animals, without the supervention of neuritis. It is, however, on the other hand, common enough as a consequence of wounds, especially of those of a lacerated character inflicted on nerve-trunks. The terminal branches of nerves do not appear to be so readily affected. As we have seen, it may result from cold ; it is also produced by exudations from the tissues through which the nerve passes, and, as Leudet[1] has shown, by the inhalation of carbonic oxide.

Diagnosis.—From neuralgia it is distinguished by the history of the case, where traumatism is a feature, by the facts that the temperature of the parts supplied by the affected nerve is always elevated, which is not the case in neuralgia, by the persistence of the pain, and by the circumstance that, except in traumatic acute neuritis, the pain is not excessive. The occurrence of paralysis, spasm, or anæsthesia, or all of these symptoms in neuritis, and their absence in neuralgia, will also serve to distinguish the one disease from the other.

From cerebral or spinal disease acute neuritis is readily diagnosticated by the absence of central symptoms and by the restricted limits of the morbid phenomena.

Prognosis.—The prognosis in cases of idiopathic acute neuritis is not unfavorable; the disease may be entirely dissipated, leaving the functions of the nerve slightly, if at all, impaired. Sometimes, however, and especially in traumatic cases, the tendency is to the continuance of the morbid process in a chronic form to the point of producing profound lesions of the nerve-tissue. Or, as Mitchell says, it may be the prime factor in the production of neuralgia, causalgia, joint-disease, and local palsies.

Morbid Anatomy and Pathology.—The lesion generally involves at once both the neurilemma and the proper nerve-elements. The vessels are enlarged, and often extravasations take place. The connective tissue is increased in amount, and an exudation of serous or sero-fibrinous fluid, with a tendency to coagulation, is formed. The tissues in the immediate vicinity of the inflamed nerve participate more or less in the morbid action.

---

[1] "Recherches sur les troubles des nerfs périphérique surtout des vaso-moteurs consécutifs à l'asphyxie par la vapeur de charbon," *Archives Générales de Médecine*, 1865.

If resolution results, these products are absorbed, and the nerve regains its normal condition; if, however, suppuration ensues, little abscesses form within the sheath of the nerve, or between its fibres; these latter become completely disorganized through granular degeneration, and eventually constitute an amorphous mass of oil-globules and *débris* contained within the neural sheath.

The pathology of neuritis, like that of other diseases, is to be studied from the stand-point of the normal physiology of the healthy nerve—and there is little to add to the remarks already made under the head of symptoms. The fact should be borne in mind that irritations applied to a nerve-centre or a nerve-trunk are more acutely felt at the points of distribution of the nerve than at the seat of the irritation. Of course, in accordance with a well-known law, irritations made to a motor or compound nerve-trunk cause spasms in the muscles to which the nerve is distributed. The first stages of inflammation constitute an irritative process. Hence the clonic contractions which are present in the early periods of the affection. But, as the morbid action proceeds, the irritability and conductivity of the nerve become abolished, and therefore the clonic spasms cease, and voluntary power in the muscles supplied by the diseased nerve is lost.

Treatment.—Mitchell, in the only case of acute neuritis of which he had the control from the beginning, enveloped the arm from above the wound to the finger-ends in bladders of ice and water; the limb was elevated above the body, and one twenty-fifth of a grain of sulphate of atropia, combined with one-quarter grain of sulphate of morphia, was given in solution every four hours or oftener if needed.

Jaccoud recommends leeches, and even cups over the course of the nerve. The latter must certainly cause suffering, and Mitchell states that even leeching, though sometimes beneficial, causes great pain, and that the leech-bites are prone to inflame.

In the cases of acute neuritis resulting from cold which have been under my charge I have obtained decided benefit from the use of the primary galvanic current of great intensity, the application being made through wet sponges drawn over the skin covering the affected nerve. Two applications, each lasting half an hour, should be made. Each one is followed by a diminution of the pain and numbness, and a lessening of the spasms of the muscles.

At the same time I have employed deep injections of sulphate of morphia combined with sufficient sulphate of atropia to counteract its unpleasant effects—one-fourth grain of the morphia with the one-sixtieth of atropia being about the doses to begin with. Two injections should be made daily. I endeavor to touch the nerve with the point of the syringe, or, failing that, to come as near to it as possible.

The good effects of this treatment are generally very evident, and a cure is ordinarily accomplished in at most a week.

In two cases of inflammation of the radial nerve, apparently resulting from long-continued exposure to cold and dampness, which have recently been under my care, I have applied the actual cautery along the whole course of the inflamed portion of the nerves. The effect was an arrest of the pain, the numbness, and the muscular spasms, and the continual relief of all the symptoms by the subsequent injections of morphia and atropia for two or three days. In both of these cases the tract of the inflamed nerve was marked by cutaneous redness.

After the disappearance of the acute symptoms, if any anæsthesia or paralysis remains, it is to be treated with the induced or primary current, as may seem most advantageous by actual experiment. I am inclined to think that both currents should be used, the primary uninterrupted for the relief of the anæsthesia and for improving the conductivity of the nerve, and the induced to the muscles for the restoration of their irritability. At the same time, passive motions, frictions with hair-gloves, and applications of hot water, are beneficial.

It must not be forgotten that in the early stages, and all through the active period of the disease, alsolute rest must be as nearly as possible secured. Every muscular contraction of a limb containing an inflamed nerve causes intense suffering, and can scarcely fail to aggravate the disease.

Constitutional treatment beyond such as may be necessary to maintain or increase the tone of the system is not ordinarily required.

CHAPTER III.

CHRONIC NEURITIS.—NEURAL SCLEROSIS.—NEURAL ATROPHY.

Chronic neuritis may result from an attack of acute neuritis; from central disease—either of the brain or spinal cord, or it may have an idiopathic origin.

Symptoms.—The symptoms vary in accordance with the physiological character of the affected nerve. If a compound nerve is the seat of the lesion, the phenomena are in the main anæsthesia, paralysis, and muscular atrophy. If a sensory nerve is the one involved, anæsthesia, and perhaps pain, is the most prominent symptom. If a nerve of special sense is affected, there is disturbance of the function of the nerve as regards the related sense, and this may be either of the character of hyperæsthesia, anæsthesia, or both. If the diseased nerve is purely motor in function, then the results are motor paralysis and muscular atrophy.

We have already had to consider to some extent the sclerosis and atrophy of spinal nerves in connection with certain diseases whose primary seat is in the spinal cord. But such nerves may be the seat of the lesion in question and may give rise to symptoms similar in some respects to those due to disease of that part of the cord with which they are in anatomical and physiological relation.

Disturbances of sensibility, usually of the nature of anæsthesia rather than hyperæsthesia and paralysis of motion strictly limited to the muscles to which the affected nerve is distributed, are the first symptoms. These become more distinctly marked as the lesion advances in its course, and eventually reach a full state of development. The electric contractility of the muscles begins to diminish early in the course of the disease, and reflex excitability is also lessened.

Secondary neuritis, when resulting from spinal lesion, may affect either the anterior or posterior root singly of one or more nerves. In such cases the eccentric disturbances are connected with motion or sensibility, as the case may be.

Chronic neuritis affecting a sensory nerve is not in general characterized by very acute pain, and this is accompanied by anæsthesia of the parts to which the nerve is distributed. As the disease advances the pain becomes less, and the anæsthesia correspondingly increases. The reflex excitability of the muscles to which the nerve is distributed is diminished for the reason that sensory impressions are not transmitted in full force along the trunk of the affected nerve, and hence are not promptly, if at all, converted into motor impulses. Neuritis as affecting the nerves of special sense does not come within the scope of this treatise.

When a purely motor nerve, as the facial, is the seat of chronic neuritis, the phenomena observed relate exclusively to motion. In the early stage there is probably clonic spasm in the muscles supplied by the nerve, but ere long paralysis takes its place—atrophy and rigidity of the muscles soon follow. Electric contractility and reflex excitability are early impaired, the latter on account of the paralysis of the muscles, and not from any retardation of the passage of sensory impressions—which of course do not travel through a motor nerve—to the central organ.

Chronic neuritis often exhibits a tendency to ascend and to involve more central trunks in the inflammatory process. Mitchell speaks of this as a constant result.

Causes.—The most common causes of chronic neuritis are the acute form of the disease and lesions of those parts of the central nervous system from which the affected nerves are derived. It also results when from any cause whatever the peripheral organs to which the nerves are distributed are prevented performing their normal functions.

Chronic neuritis may originate primarily from wounds and injuries without necessarily being preceded by an acute attack.

Cold may be a factor in its causation, and, in conjunction with damp, probably produces most of the idiopathic cases.

Syphilis, undoubtedly, may give rise to chronic neuritis. I am quite sure that several cases having this origin have been under my care—and the fact is admitted by Lagneau,[1] Buzzard,[2] and others.

Diagnosis.—Chronic neuritis is distinguished from progressive muscular atrophy mainly by the circumstance that the paralysis precedes the atrophy, the latter being a secondary condition, while in progressive muscular atrophy it is the primary essential phenomena. The presence of pain, the absence of fibrillary contractions, the impairment of the electric contractility, and the existence of anæsthesia, will further serve to make the diagnosis exact. Moreover, the clinical history cannot fail to add to the distinctive features of the two affections.

From neuralgia it is diagnosticated by symptoms and characteristics to which attention has been drawn in the immediately preceding chapter.

Prognosis.—Mitchell regards the prognosis of chronic or subacute neuritis as grave in proportion to the length of nerve involved, and the extent to which the morbid process has traveled in a central direction. His opinion is based rather upon the traumatic variety of the disease than the idiopathic. To the opinion expressed by him I would add that the chronic neuritis which results from central lesions is particularly hopeless. That due to syphilis is not generally unamenable to treatment.

Morbid Anatomy and Pathology.—The process which characterizes chronic neuritis is not essentially different from that which marks chronic inflammatory action in the white tissue of the spinal cord. It consists in a hyperplasia of the neuroglia and a contemporaneous atrophy of the nerve-tubes. The white substance of Schwann undergoes fatty degeneration, and the nerve-tubes remain as dense fibrous cords. The morbid action, therefore, takes on the features of sclerosis, even to the production of the characteristic gray coloration.

The main points in the pathology of the affection have already been mentioned, and need not, therefore, be again considered.

Treatment.—Except in the chronic neuritis due to syphilitic infection there is not much to do in the way of internal medication. In this form the iodide of potassium, given in gradually-increasing doses, as previously recommended for chronic basilar meningitis of like origin, is necessary, and will often, if the disease has not advanced too far, effect a cure. In all forms the primary galvanic current, of as great a degree of intensity as the patient can bear, should be applied to the cutaneous surface over the affected nerve. The conducting power of the skin should be increased by wetting it, and the electrodes should be wet sponges. If the nerve is so situated as not to be acted upon directly, the current should be so applied as to affect it secondarily. For instance, the third nerve may be subjected to the galvanic stimulus by one pole being placed over the closed eye, and the other on the nape of the neck. The same process answers for the optic, fourth, and sixth nerves. Care should be taken to use a current, in such cases, of low tension; and, in all applications of the primary current to the face, this caution should be remembered.

---

[1] "Maladies syphilitiques du système nerveux," Paris, 1860, p. [illegible], et seq.

[2] "Clinical Aspects of Syphilitic Nerve Affections," London, 1874, p. [illegible]

For the paralyzed muscles, the procedure recommended in the previous chapter of using either the galvanic or faradaic current, or both, as the case may appear to demand, is equally applicable to chronic neuritis.

Hypodermic injections of strychnia are useful. The initial dose may be about one twenty-fourth of a grain, and this may be gradually increased. The injection should be made at a point as near to the affected nerve as may be possible and proper.

CHAPTER IV.

TUMORS OF NERVES.

THE nerves, like the brain and spinal cord, are subject to morbid growths; but very little is known of them either clinically or anatomically. Gummy tumors, syphilitic in origin, and giving rise to symptoms not essentially different from those just described as characterizing chronic neuritis, are known to exist. The following case from Buzzard[1] was probably one of the kind in question:

"A laborer, aged thirty-one, applied in February, 1873, with paralysis of the right lower extremity of two and a half years' standing. The limb was greatly wasted, and the foot could not be moved at all. It seemed that on getting up one morning he found his foot useless. No pain had preceded or followed the attack, and he had not been ill. Excitability to the induced current was lost in all the muscles below the knee, and very much diminished in the muscles at the back of the thigh, while it remained good in those on the anterior aspect of the thigh, in which also he retained voluntary power. The sensibility of the skin in parts corresponding to the paralyzed muscles was greatly diminished. There was no increased excitability to the intermitted constant current. The seat of paralysis corresponded completely with the distribution of the great sciatic nerve. There was no impairment of the functions of the bladder, nor of the other leg. It appeared evident that there was a lesion of the sciatic nerve alone. Although he positively denied any syphilitic infection, the existence of a very uglylooking sore on the right leg (suggestive of a gummatous ulceration), which he had had for three months, made it likely that there had also been a gumma of the sciatic nerve, and he was accordingly ordered iodide and mercury. Under this treatment the sore rapidly healed, he gained a certain amount of power in the leg, and he described himself as feeling more than usually well in his general health, but in April he ceased to attend, so that I am unable to give the sequel of his case."

---

[1] *Op. cit.*, p. 112.

Virchow,[1] while admitting that the nerves may be the seat of gummy tumors, declares that those most frequently affected are the optic, the olfactory, the third, fourth, fifth, and sixth. He refers to a case cited by Zambaco, in which the crural was apparently the seat of a gummy tumor.

Besides the gummy tumor, nerves are subject to cancerous tumors, to myxomata, and to various forms of neuromata, among which the painful tubercule is specially to be mentioned. These latter are small, and generally situated just under the skin.

The treatment of neurotic tumors is not medical except for those which are of syphilitic origin. For these the iodide of potassium and mercury are the remedies which are indicated. All others require excision.

CHAPTER V.

NEURAL PARALYSIS.

FACIAL PARALYSIS.

Paralysis of the facial nerve has already been considered as a symptom of several central lesions, but it may exist as an affection of altogether peripheral origin. As such, it is often known as Bell's paralysis, on account of its real nature having been first clearly pointed out by Sir Charles Bell. The nerve in question, the facial or portio dura of the seventh pair, was formerly regarded as one of sensation, and, in accordance with this view, was often divided for neuralgia. The experiments of Bell and Magendie established the fact of its being entirely a nerve of motion.

Symptoms.—The facial nerve is distributed to nearly every muscle of the face. Its paralysis therefore causes such decided change of expression as to be readily recognizable. The most marked phenomenon, and one which is of importance in the diagnosis, is the inability to close the eye of the affected side. This is due to the fact that the orbicularis palpebrarum has lost its contractile power, while the levator palpebræ superioris, not supplied by the facial, but by the third nerve, is not paralyzed, and keeps the upper lid elevated. In consequence of this condition, the eye is constantly exposed to the action of the atmosphere, and to contact with extraneous substances. The patient cannot wink, and thus the tears, not being distributed over the surface of the eyeball or carried off by the nasal duct—the tensor tarsi also being paralyzed—run over the lower lid, and scald the cheek. From this inability to wink, dust and other small particles of matter are not removed, and hence considerable irritation is produced. Exposure to strong light or to wind adds to the inconvenience. Comparative comfort may be obtained by the patient frequently closing the eye with the finger, or by keeping the lids together with a piece of adhesive plaster.

---

[1] "Pathologie des tumeurs," French edition, tome ii., Paris, 1869, p. 454.

The next most prominent group of symptoms is due to the loss of power in one lateral half of the orbicularis oris. As a consequence, the patient cannot purse up the mouth on that side, as in the act of whistling or spitting. From this loss of tonicity the saliva is not retained on the affected side of the mouth, but runs out over the lip, to the great annoyance of the patient.

The muscles of mastication, the masseter, temporal, and external and internal pterygoid, are supplied by the third branch of the fifth pair of nerves, and hence the ability to chew is not impaired. The buccinator, the function of which, in conjunction with the tongue, is to press the alimentary bolus against the jaws, and thus keep it submitted to their action, is supplied by the facial, and hence its office is not performed. The food consequently accumulates between the jaws and the cheek, and it must be continually removed by the finger.

The muscles which expand the face, as in the action of laughing or smiling, are supplied by the facial, and their paralysis destroys the normal equilibrium, and hence the face is drawn toward the sound side. This loss of antagonism is most evident when the patient opens his mouth, and particularly when he laughs or smiles, for the paralyzed muscles, the zygomatici, and the risorius, are incapable of responding to the emotion, while those on the sound side contract vigorously.

The paralysis of the occipito-frontalis and of the corrugator supercilii prevents the raising of the eyebrows, or frowning, and obliterates all wrinkles from the brow. As Romberg remarks, there is no better cosmetic for elderly ladies than facial paralysis ("für alte Frauen kein wirksameres Cosmeticum existirt").

Among other symptoms, it is noticed that the ala nasi is depressed, and does not expand as air is drawn in through the nostril, and that the articulation, especially of words containing labials, is very indistinct.

The expression of one side of the face is therefore destroyed; it is a complete blank, incapable of responding to any emotion, and unable to execute those movements which in the normal condition are performed by its muscles.

Such are the obvious and superficial symptoms of an ordinary attack of unilateral facial paralysis. For the full understanding of other important phenomena, a few words in relation to the anatomy and physiology of the nerve are necessary.

The facial nerve takes its origin from the posterior border of the pons Varolii and the lateral tract of the medulla oblongata. Some of its fibres of origin may be traced to the floor of the fourth ventricle, and even to the lateral columns of the spinal cord. A knowledge of its course and connections enables us to determine with a good deal of accuracy the seat of the lesion by which it is paralyzed, and thus we have an important element in making a prognosis.

From its point of apparent origin the facial passes forward and outward, resting on the crus cerebelli, and leaves the cranial cavity by entering the internal auditory meatus with the auditory nerve. It next enters the aqueductus Fallopii, and, passing through its whole length, makes its exit from the skull by the stylo-mastoid foramen; while in the aqueductus Fallopii it gives off three branches, the two superficial petrosal nerves, and the chorda tympani. The great superficial petrosal passes to Meckel's ganglion, and through this supplies the levator palati and the azygos uvulæ muscles; the small superficial petrosal—regarded by some as a branch of the glosso-pharyngeal, though communicating with the facial—runs to the otic ganglion which supplies the tensor-palati and tensor-tympani muscles, and also, according to Bernard, presides over the secretion of the parotid gland, through the auriculo-temporal nerve; the chorda tympani goes to join the gustatory branch of the fifth, and is in part distributed with this to the tongue, but another portion of its fibres enters the submaxillary ganglion which presides over the function of the submaxillary gland.

With this brief *résumé* of the anatomical and physiological points of the facial nerve, we are prepared to study other symptoms to which I have not as yet alluded; for, in the account given, I have simply considered the phenomena of facial paralysis when the lesion is situated on the distal side of the stylo-mastoid foramen. But the nerve may be affected farther back, and, though in such a case we have the symptoms already described, there are others which vary according to the seat of the disease.

Thus, if the morbid process is in action above the origin of the chorda tympani, but below that of the petrosal nerves, the patient will experience a diminution but not a complete abolition of the sense of taste upon the corresponding side of the tongue. This fact led to the supposition that the chorda tympani was a sensitive nerve, but the experiments of Bernard and others have clearly shown that it is an efferent nerve, conveying influence from the brain, not to it. One of its actions is to increase the flow of submaxillary saliva. In addition, it supplies the lingualis muscle, and probably erects the papillæ of the tongue, and modifies the circulation of this organ. When, therefore, a lesion of the facial exists above the origin of the chorda tympani, the sense of taste on that side is lessened because the dryness of the mouth prevents the ready solution of the sapid substance. The difficulty is augmented through the non-erection of the papillæ, and perhaps, also, by the change which has ensued in the circulation. This diminution of the sense of taste therefore shows that the lesion is seated on the central side of the origin of the chorda tympani nerve.

Again, if the lesion be situated behind the gangliform enlargement, from which the petrosal nerves arise, but anterior to the meatus internus, we have, of course, all the symptoms mentioned, and in addition those due to paralysis of the petrosal. One of them is the depression of the palatine arch on the affected side; it hangs lower than the opposite one, and its edge is nearly straight instead of curved. This condition results from paralysis of the levator-palati muscle, which, as we have seen, is supplied by the great petrosal through Meckel's ganglion. One of the two little muscles of the uvula being powerless, the other draws the uvula into a bow shape, with the concavity toward the sound side. The uvula and the velum are also pulled *en masse* toward the sound side by the action of the tensor palati, the other being paralyzed through the implication of the small petrosal nerve. The connection of the small petrosal through the otic ganglion with the parotid gland causes a diminution of the secretion of this gland when the lesion of the facial is in the situation described.

Acuteness of hearing on the paralyzed side is sometimes observed. This is accounted for by Landouzy,[1] on the ground of the paralysis of the tensor-tympani muscle, which, as we have seen, is supplied by the otic ganglion, but Brown-Séquard attributes it to hyperæsthesia of the acoustic nerve from vaso-motor spasm.

This last category of symptoms, therefore, indicates the seat of the lesion to be at or behind the gangliform enlargement.

When the lesion is within the cranium, we have all the symptoms mentioned, but they are complicated with others indicative of derangements of other nerves, or of cerebral disease. These have already been considered under other heads.

In the foregoing account of facial paralysis, the unilateral form, which is by far the most common, has alone been considered, but both nerves may be paralyzed, producing what is called double facial paralysis, or facial diplegia. The condition has been well described, among others, by Wachsmuth,[2] and by Pierreson,[3] the latter of whom has collected twenty-eight cases as the basis of his memoir. Both sides may be paralyzed simultaneously, in which instance the disease is probably central, or one may follow the other. In either case, the face presents

[1] "De l'altération de l'ouïe dans la paralysie faciale," *Gazette Médicale*, Paris, 1851.

[2] "Ueber progressive Bulbar-Paralyse und die Diplegia facialis," Dorpat, 1864.

[3] "De la diplegie faciale," *Archives Générales Médecine*, Août, 1867, p. 139.

a complete want of expression, and the symptoms previously mentioned are duplicated in full. Two excellent representations of the affection are given in the report of a case by Mr. Wright.[1] Only one case has come under my observation. It was of long standing and incurable. I lost sight of the patient before I could have his photograph taken.

Causes.—Cold is a prominent cause of facial paralysis. It is most apt to induce that form of the disease in which the lesion is external to the temporal bone. Exposure to intense cold, especially when the wind was blowing, has caused several cases in my experience. The patient has gone to bed feeling pretty well, and has awakened with one side of the face paralyzed.

Rheumatic inflammation, occurring in the course of the nerve, may also induce facial paralysis, as may likewise tumors of the parotid gland, or other cause capable of making pressure on the nerve. I have seen several cases which had resulted from sleeping with the closed hand under the face; and it may occur in new-born children, as the result of pressure by the forceps. Wounds and injuries of other kinds may, of course, produce it.

Within the temporal bone, facial paralysis may result from tumors, from periostitis, from caries of the petrous portion of the temporal bone, from disease of the middle ear, from hæmorrhage into the aqueductus Fallopii, and from fractures of the temporal bone.

Within the cranium it may be caused by disease of the pons Varolii, or of the medulla oblongata, by atrophy of the nerve, by tumors, and as the consequence of injury. These latter do not, however, demand our notice, as they have already been considered in other connections.

Diagnosis.—Facial paralysis is distinguished from glosso-labio-laryngeal paralysis, by the facts that in the latter the symptoms affect only the lower part of the face, and that they are accompanied by paralysis of the tongue and of the muscles of deglutition. From the facial paralysis of hemiplegia it is diagnosticated by the marked circumstance that, in the latter disorder, the patient can close the eye, while in the former it remains wide open. There are no other affections with which facial paralysis can be confounded, if the slightest attention be given to its symptoms.

Prognosis.—The prognosis varies according to the seat and the cause of the lesion, and the duration of the paralysis. If this latter is due to cerebral or intra-cranial lesion, or to disease existing within the aqueductus Fallopii, the prospect of cure is remote. But, if the lesion exists outside of the skull, and is capable of removal, or if the paralysis be the result of exposure to cold, or subjection to pressure, and if the electric contractility of the muscles be not destroyed, the case, under suitable treatment, will probably terminate favorably. By electric contractility, I do not mean the ability to respond to the excitation of the induced current, for this is lost at an early period in the majority of cases, but to contract upon the application of as strong a primary current as can with safety be applied to the face.

---

[1] "Notes of a Case of Double Facial Palsy," *British Medical Journal*, 1869, p. 184.

In deep-seated lesions, if a clinical history of syphilis can be made out, the prognosis becomes more favorable.

If the affection has lasted a long time, and if contractions of the paralyzed muscles from atrophy have taken place, the probability of recovery is very slight, even if there is some glimmering of electro-contractility.

Morbid Anatomy and Pathology.—When facial paralysis results from cold, it may be from consequent neuritis, or from inflammation excited in contiguous parts. In the latter case lymph is effused and pressure is exerted upon the nerve. Most of the other causes act by the pressure they make on the nerve, and, though, as in the case of sleeping with the fist under the face, the action may not be long continued, the consequence is very lasting. The effects of pressure upon a nerve are experienced when we sit too long in one position, so as to compress the sciatic nerve, or when persons go to sleep with one arm thrown over the back of the chair on which they are sitting. The axillary plexus is compressed, and paralysis, more or less complete, of the muscles supplied by it, is the result. Several such cases have come under my observation, and the resulting paralysis is generally most difficult to remove.

The fact that in the affection now under notice the orbicularis palpebrarum is paralyzed, while in facial paralysis, symptomatic of cerebral disease, such as hæmorrhage, it escapes, is to be explained by the circumstance that in the latter disease all the fibres of origin of the nerve are not involved, while in the former the whole trunk of the nerve is subjected to the morbid process, and hence all the muscles which it supplies are paralyzed.

Treatment.—The indications are: to remove the cause if possible; to put the nerve under the best possible conditions for regaining its lost power; and to preserve the organic integrity and irritability of the muscles till this can take place. When there is reason to suspect the existence of a syphilitic taint, and the growth of exostoses of syphilitic character in the aqueductus Fallopii, the iodide of potassium with the bichloride of mercury should be given, according to the formula on page 308. In several cases I have succeeded in effecting cures by this treatment, conjoined with electricity, when the latter by itself had produced no improvement, or the iodide may be given alone in gradually-increasing doses, as recommended for chronic basilar meningitis.

For the restoration of the nerve-function, we can do little beyond securing healthy nutrition of the general system, by the use of proper hygiene and tonics. Among the latter, strychnia is especially useful.

It should be employed persistently and in gradually increasing doses, till some evidence of its physiological action is obtained. For this purpose I make use of a solution of the sulphate of strychnia in the proportion of one grain to the ounce of water. Every ten minims of such a solution contain $\frac{1}{48}$ of a grain of the medicine. Generally I begin with ten minims of this solution three times a day for the first day; the next day eleven minims are given three times; the next twelve, and so on, till the patient experiences a sensation of cramp or rigidity in the legs, or in muscles of the back of the neck or of the jaw. Usually the cramp is first felt in the calves of the legs. The further administration is now stopped, and, if necessary, on the following day the solution is given as before, in doses of ten minims, and the doses are again run up to the extent of producing the muscular cramp. As illustrative of the action of this method, I cite the following case from my note-book. It is one of twenty-eight others in which the practice referred to was adopted.

Miss S., in coming from Newark to New York, on the evening of January 5, 1878, opened the car window over the seat on which she sat. She experienced no inconvenience till the following morning, when on awaking she found that the left side of the face was paralyzed. On the 7th she came under my observation. Examination showed that not only were all the muscles of the face supplied by the facial nerve paralyzed, but that there was a diminution of the sense of taste on the side of the tongue corresponding to the paralyzed side of the face, that the left palatine arch was straighter and lower than the right, and that the uvula was concave toward the paralyzed side, while this organ and the velum were drawn over toward the sound side. These phenomena indicated that the lesion or morbid process was situated behind the gangliform enlargement.

I at once began the administration of the strychnia, according to the formula just given, placed the hook (to be more specifically mentioned directly) in the angle of the mouth on the left side, and advised the use of the faradaic current for a few minutes every alternate day. On the tenth day, while taking the $\frac{1}{24}$ grain of the strychnia, she felt a little rigidity of the muscles of the calves of the legs. It was so slight, however, that I advised the continuance of the increasing doses. But even now the improvement was evident. She could close the eye of the affected side, elevate and corrugate the brows, and slightly retract the angle of the mouth. When she laughed, however, the right angle of the mouth was retracted much farther than the left.

But soon after taking the third dose of twenty-one minims, on the following day, she experienced very decided cramps in both legs, which, however, passed off in less than half an hour. On the next morning I saw her. The action of the facial muscles was, so far as I could see, equal on both sides. There was no relapse.[1]

---

[1] "On an Improved Method of Treating Facial Paralysis," *St. Louis Clinical Record*, May, 1878.

I have never found blisters or liniments to be of the slightest service.

The third indication is to be met by passive exercise, such as can be produced by pinching and kneading the muscles, and, above all, by the persistent use of electricity. Without this latter agent facial paralysis cannot be cured.

If the induced current will cause the muscles to contract, it should be employed. One pole is placed over the nerve at its exit from the stylo-mastoid foramen, and the muscles of the paralyzed side are separately excited by the other. A *séance* should last about fifteen minutes, and should be repeated every alternate day, or every day in bad cases.

If the induced current will not cause contractions, the primary interrupted current should be used for the purpose. Care should be taken not to employ a current of too great a degree of intensity, as serious consequences have resulted to the vision by neglect of this precaution. As a rule, fifteen Smee's cells will be sufficient; but, if the current be passed through a column of water before it reaches the face, a larger number of cells may be used with safety. Means must be taken to interrupt the current, as contractions are only produced when the circuit is closed and opened. When the primary current has been employed for a few weeks, it will generally be found that the induced will cause the muscles to contract, in which case it should be substituted.

The first muscle to recover power is usually the orbicularis palpebrarum, but several weeks, and sometimes months, are requisite to bring about a complete cure.

As an additional measure, which is serviceable in restoring the muscles of the mouth, the use of a very simple apparatus calculated to relax them is to be recommended. It consists of a hook made of hard rubber or whalebone, or some other suitable substance, which is caught into the angle of the mouth on the paralyzed side, and then attached to the corresponding ear by means of an elastic band. The first to use such an appliance, so far as my knowledge extends, was Dr. William Detmold,[1] of this city, who, in an old case of facial paralysis, obtained great benefit from its application. His apparatus consisted of a piece of silver wire bent into a hook at one end, for the angle of the mouth, and then bent again at the other end, and carried over the top of the ear somewhat after the manner of a pair of spectacles. The elastic band, such as is used to keep letters together, is, I think, an improvement.

PARALYSIS OF THE THIRD NERVE—MOTOR OCULI.

Symptoms.—The motor-oculi nerve which supplies the upper eyelid, the superior, inferior, and internal recti muscles, the inferior oblique, and indirectly, through the ophthalmic ganglion, the circular, or constricting fibres of the iris, has already been considered in its central pathological relations. It is, however, the seat of peripheral disease, either intrinsic or as a consequence of lesion of the contiguous tissues. When the trunk of the nerve is the seat of disease or subjected to pressure, the symptoms consist of ptosis or a drooping of the upper eyelid, external strabismus from the action of the uncompensated external rectus muscle, and dilatation of the pupil from the uncompensated action of the dilator pupilaris muscle.

---

[1] "Facial Paralysis treated by a New Method," *New York Medical Journal*, May, 1873, p. 491.

The patient, therefore, presents a remarkable appearance. The upper eyelid hangs down over the cornea, almost but not quite, in extreme cases touching the lower lid; the eyeball is turned outward, and, from the destruction of the parallelism of the axes, double vision is produced and the pupil is more or less widely dilated and insensible to the stimulus of light.

The external rectus and the superior oblique of all the extrinsic muscles of the eyeball remain unparalyzed, but as all the antagonizing muscles are powerless, they are in a state of tonic contraction, and the mobility of the eye is hence destroyed.

Generally, however, in peripheral paralysis of the third nerve, the muscles most frequently affected are the levator palpebræ superioris, or the internal rectus, or both; and the branches supplying these parts are therefore alone involved.

Cases of the kind are not uncommon. An interesting case came under my observation not long since in consultation with Drs. T. B. Sterling and T. C. Finnell. Recovery took place under suitable treatment, but, some six months afterward, the patient, a boy, about twelve years old, was brought to me by his mother on account of a recurrence. A cure was again easily effected. In this case the disease was apparently the result of reflex action from the stomach. The regulation of the diet, the internal use of strychnia, and the application of the faradaic current to the closed eye in both instances relieved the condition in a couple of weeks.

Causes.—Peripheral paralysis of the third nerve may be induced by syphilitic or other tumors compressing the nerve, by rheumatic exudations along its course acting in like manner, by blows upon the eyeball, or by other injuries; by currents of cold air blowing upon the eye, or by reflex irritations, such as indigestible food or worms in the alimentary canal. The two latter are especially active causes in children.

Diagnosis.—From central disease peripheral paralysis of the motor oculi nerve is readily distinguished by the absence of "head-symptoms."

Prognosis.—This depends very much upon the cause. If the paralysis results from pressure it will continue so long as the factor remains in operation. Syphilitic tumors are more readily removed by constitutional treatment than any others. These latter may, however, in some cases, be gotten rid of by surgical operation. When the affection is induced by wounds or injuries, recovery is probable unless the structure of the nerve has been seriously impaired. When it is caused by cold, rheumatic exudations, or reflex irritations, recovery is the rule.

The Morbid Anatomy and Pathology scarcely call for any additional remarks; and the **Treatment** is to be conducted upon the same principles as those laid down for facial paralysis. It is, however, worthy of special mention that the tension may be advantageously taken from the muscle of the upper eyelid by the use of a thin piece of India-rubber, which is to be attached to the lid and to the skin above it by collodion, as recommended by Dr. John Van Bibber, of Baltimore.

Division of the external rectus may, in old cases, be necessary for the obviation of the strabismus.

Paralysis of the sixth or abducens nerve, by which the eye, owing to the loss of power in the external rectus and the uncompensated action of the internal rectus, is turned inward and double vision produced, has a like clinical history, and is to be treated upon like principles.

The same may be said *mutatis mutandis* of other peripheral paralyses, as, for instance, of the muscles of the larynx, of the deltoid, and of the muscles supplied by the radial nerve.

Relative to this latter, the *radial*, M. Panas[1] has shown that the paralysis to which it is liable is not, as generally supposed, the result of cold, but of slight pressure, to which it is often subjected, and M. Desplats[2] adduces additional arguments in support of this view.

The latter cites the following case, the details of which were given to him orally by MM. Panas and Raynaud:

In 1874 there was in the wards of M. Raynaud, at the Lariboisiére, a patient affected with phthisis, and who was suddenly one night taken with paralysis of the left radial nerve. At the morning visit the fact was noticed and the cause sought for. The patient habitually slept on the right side, and the idea of compression was therefore dismissed, the paralysis being attributed to cold. But a neighboring patient stated that he had seen the paralyzed man sleeping with his left arm lying on the table by the side of his bed, and his head resting on it. This satisfactorily accounted for the paralysis. In the course of a few days it was cured by electrization. But a few days afterward the patient died.

The post-mortem examination of the nerve was made with great care by MM. Panas and Raynaud, and they both remarked that at the point where the compression had been made the nerve was of a very decided ochrey color. The portion thought to be injured was examined in the laboratory of the College of France, but no further alteration was detected. This was not remarkable, as the functions of the nerve had been restored for several days.

---

[1] "De la paralysie réputée rheumatismale du nerf radial," *Archives Générales de Médecine*, 1873, p. 657.

[2] "Des paralysies péripheriques," Paris. 1875 p. 61.

CHAPTER VI.

NEURAL SPASM.

There are two affections which may be taken as the types of peripheral spasm in general: these are spasm of the facial muscles—the mimic or histrionic spasm of Romberg, the convulsive tic of the French—and torticollis, or the spasm in the muscles of the neck supplied by the spinal accessory nerve.

FACIAL SPASM.

The spasms in the disease under notice may be either clonic or tonic, the former being by far the more common. In the clonic form, the muscles of the face, or a portion of them, generally on one side, are suddenly and violently contracted, and as suddenly relaxed. Sometimes, the angle of the mouth is drawn back; again, the upper lip and the alæ of the nose are elevated; and again, the spasm affects the orbicularis palpebrarum. In a case formerly under my charge, occurring in a gentleman from Rahway, New Jersey, both orbicularis muscles were affected with clonic and tonic spasms, the eyes sometimes being closed for several minutes at a time.

The spasms come on in paroxysms which are of variable duration. I have seen them last continuously for over an hour. Generally, they continue from a few seconds to one or two minutes, and are repeated at intervals of about the same time. They may generally be excited by emotional disturbance of any kind; by muscular exertion, by a current of wind, or other cause capable of exciting reflex actions. In the case above referred to, they are always induced by walking. They can be made to cease by pressure upon the facial nerve at various points, and they are generally arrested by powerful mental occupation and by sleep.

In the tonic form of the affection the spasm persists, and causes more or less distortion of the face. It interferes with articulation, mastication, and especially with emotional expression.

The tendency is for either form to become habitual, and hence to be difficult of cure.

Causes.—Cold is a common cause, as are also wounds and injuries, and carious teeth. I have seen two cases recently, from this last-named influence.

The **Diagnosis** calls for no special consideration, and the **Prognosis** depends very much upon the duration. Generally, it is unfavorable.

There are no facts bearing on the **Morbid Anatomy**, and the **Pathology** is to be explained by the principle of reflex excitation which, in this case, probably takes place through the intermediation of the fifth pair; by reference to the facts ascertained by the experiments of Fritsch and Hitzig, Nothnagel, Ferrier, Bartholow, and myself; or by the theory of irritation existing somewhere in the course of the facial nerve. The latter is probably the most common condition. The analogy with chorea is very great.

Treatment.—Of fourteen cases that have been under my charge, six were cured. The means which I have found most useful are, daily hypodermic injections of a mixture in water of five drops of Fowler's solution, and the one-fiftieth of a grain of atropia, and the daily use of the constant primary current to the facial nerve and the convulsed muscles.

In several cases I have obtained good results from permanent pressure over the facial nerve. The gentleman previously referred to had had, at my suggestion, a steel spring constructed, which terminated in two pads, and which he wore over the head in such a way as to compress the facial nerves at their exit from the stylo-mastoid foramen. While he wore it he had no spasms, but he was unable to endure the pressure longer than a couple of hours.

In one case, that of a young gentleman, from the interior of this State, in whom the disease had lasted about a year, a permanent cure was produced within a month by the use of the bromide of zinc in gradually-increasing doses, as recommended for convulsive tremor, and the employment of the primary galvanic current to the skin over the facial nerve and affected muscles.

Division of the affected muscles has been practised with very moderate success.

TORTICOLLIS.

In this disease the spasms—which, as in the corresponding affection of the face, may be either clonic or tonic—occupy the sterno-cleido-mastoid, the trapezius, the rhomboid, and the levator-anguli scapulæ, separately or collectively. The movements of the head in the clonic form depend upon the seat of the spasms, the action being in the direction of the tractile force of the affected muscles. Sometimes the contractions are very rapid, and again they are slow and regular; as in facial spasm, they are aggravated by emotional excitement or physical exertion. They cease during engrossing mental occupation, and during sleep. Occasionally both sides are affected.

The reverse as regards facial spasm, the tonic form, is much the more common, and it is to it that the term torticollis is usually applied by surgical writers. The sterno-cleido-mastoid is generally its exclusive seat. The contraction is often accompanied by pain.

Causes.—The etiology is not essentially different from that of facial spasm.

Diagnosis.—There is no difficulty about the diagnosis of the clonic variety. The tonic form is, however, liable to be confounded with a similar affection so far as appearances and consequences go, which is a veritable myositis, but which is not an affection of the nervous system. The transitory character of the latter affection and the severe pains are sufficient diagnostic marks.

Prognosis.—The prospect of recovery from the clonic form is very remote. Of ten cases that I have had under my charge, four only were cured.

Of the **Morbid Anatomy**, or of the **Pathology**, nothing is known, though the disease may be regarded as similar in its pathology to facial spasm.

Treatment.—I have made use of every remedy, in the clonic form, which could in my opinion be of service. Iron, belladonna, arsenic, morphia, chloral, chloroform, ether, bromide of potassium, strychnia, zinc, and many other medicines, have all failed. In one case I administered morphia hypodermically in gradually-increasing doses, till at last two grains were given twice a day, but without any permanent effect. I have divided the muscles in four cases without benefit. In one of them I cut both sterno-cleido-mastoids, the left trapezius at its insertion into the occipital bone, the left levator-anguli scapulæ, and finally, with the concurrence of my friend Prof. Markoe, the left complexus. But as soon as one muscle was cut another became affected, and, after the division of the complexus, the expectation of obtaining a cure by myotomy was given up. The patient, a lady, from the South, remained affected for several years, but when I last heard from her she had greatly improved, the disease having apparently exhausted its power.

Electricity in any form has never cured a case in my hands, though I have employed it steadily, for weeks at a time, both as the primary and induced currents. The induced current, however, may be used with advantage to the muscle of the opposite side as a means of improving its nutrition and strength.

In two of the successful cases, many means were tried without success. In one, that of a young man from Newark, in addition to other means, I divided the right sterno-cleido-mastoid muscle twice, and it was afterward cut by my friend Prof. Sayre. All the operations were unsuccessful, although, as in the other cases, an apparatus was worn to prevent the too rapid union of the muscle. This patient was finally cured with large doses of the bromide of potassium.

In another case, that of a lady of this city, every means used failed, till I tried the oxide of zinc; she began with doses of two grains three times a day, which were gradually increased. When she reached fifteen grains at a dose, the spasms ceased and did not return. The bromide of zinc is preferable to any other form of the remedy.

For the tonic variety, myotomy is the proper remedy, and it is generally successful if a suitable apparatus be subsequently worn.

Atropia administered hypodermically, as recommended by Da Costa,[1] has been of great service in two cases, but in both it was used in conjunction with the bromide of zinc and faradization of the non-spasmic muscles. I began with the one hundred-and-twentieth of a grain at a single daily injection, and gradually increased to the one twenty-fifth. In one case, a lady about forty years of age, recovery took place in five weeks; and the other, a young man twenty years of age, in about a month.

My experience leads me to the conclusion that division of the affected muscles, even if not immediately successful, is an important adjunct in the treatment, and it may with this object be repeated several times. The effect is to give predominance to the opposing muscles, indirectly doing what faradization is intended to accomplish. This is especially true of the sterno-mastoid muscle, the usual seat of the morbid action.

In an interesting article, Dr. John W. Ogle[2] discusses the question of the propriety of division of the sterno-mastoid and spinal accessory nerves. Thus far no great success appears to have followed the operation.

CHAPTER VII.

NEURAL ANÆSTHESIA.

ALMOST any part of the body may be deprived of sensation from causes acting on the peripheral nerves. One of the most familiar examples of this fact is the anæsthesia produced in the foot and leg by pressure on the sciatic nerve in the act of sitting too long in one position; another is the loss of sensibility produced in the hand and arm by pressure on the ulnar nerve as it passes over the elbow.

Anæsthesia originating from cerebral, spinal, or cerebro-spinal causes, has already been considered, and the present remarks will be strictly limited to the anæsthesia of peripheral origin.

ANÆSTHESIA OF CUTANEOUS NERVES.

Symptoms.—The symptoms of anæsthesia from peripheral causes do not vary materially from those which result from central lesions. They consist of the various sensations of numbness, such as tingling, "pins and needles," a feeling as if ants are crawling over the skin, water trickling over it, and, in complete cases, of absolute abolition of sensibility. The conducting power of the nerve may be impaired in so much as only to cause a retardation of the velocity of excitations, and thus an impression made on the terminal extremities of a nerve is not felt for a much longer time than would normally be the case. Peripheral anæsthesia may be accompanied with disorders of nutrition from irregularity of blood-supply. One form of the affection, of which I have seen several examples, and which probably owes its complication to vaso-motor spasm, is characterized by unnatural whiteness and shrinking of the skin, usually in the hands. If an incision be made, little or no blood escapes. In a young lady from Savannah, who was under my charge a short time since, this condition existed to an extreme degree, but disappeared with the removal of the anæsthesia. In former times, the test for the identification of witches consisted in finding a spot which could be pricked with a sharp instrument without the suspected person feeling the wound and without blood flowing. As many supposed witches were of highly-nervous temperaments, it is probable there were parts of their bodies into which pins could be stuck without causing pain or loss of blood, owing to the existence of vaso-motor spasm such at that mentioned. Anæsthesia of peripheral origin in the cutaneous nerves is sometimes accompanied by more or less loss of power, but in such cases the larger branches of the nerves must necessarily be involved.

---

[1] "Pennsylvania Hospital Reports," 1868, p. 392.

[2] "Clonic Spasmodic Contraction of the Muscles of the Neck possibly having its Origin in some Affection of the Contents of the Spinal Canal," "London Clinical Society's Reports," vol. vi.

In cutaneous anæsthesia there is always a diminution of temperature in the affected part, and this is readily detected by comparison with the corresponding healthy part by means of Dr. Lombard's thermo-electric apparatus.

Sensations are sometimes perverted. Hot bodies applied to the skin may feel cold, and cold bodies hot. Again there is usually a loss of the power to discriminate differences of temperature even when they are considerable.

The ability to distinguish slight differences in weights is usually lost, from the fact that the sense of pressure upon the skin is diminished or abolished. If, however, the difference be great, the muscles will detect it independently of the sense of cutaneous pressure. The sense of touch may remain, and that of pain be abolished—or *vice versa*. I have repeatedly observed; in cases in which I have applied the ether-spray to the skin for the purpose of preventing the pain of the actual cautery, that the patient has felt the pressure of the white-hot instrument, but has been absolutely insensible to the burning.

The æsthesiometer affords a ready means of determining the comparative and absolute loss of sensation in an anæsthetic region, and will often be of great service in the formation of a diagnosis between various subjective feelings and true insensibility.

There are certain diseases of the skin which are accompanied with anæsthesia. The principal of these are lepra anæsthetica, alopecia areata, pellagra, acrodynia, and Norwegian leprosy. In such instances the cutaneous insensibility is probably not the primary condition, but is secondary to the special skin-disorder.[1] At the same time the symmetrical character of some of these affections is, by some authorities, regarded as evidence of their dependence upon derangement of the nervous system.

Causes.—Peripheral cutaneous anæsthesia may be produced by a variety of causes. Among the chief are wounds and injuries of various kinds, whereby the nerve is divided or its conducting power impaired; pressure such as that caused by tumors, tight clothing, or accidental influences; rheumatism; exposure to intense cold, such as that produced by ice and salt or the ether-spray; the action of certain drugs, such as aconite locally applied; frequent immersion of the hands in hot water impregnated with soap, as in washer-women; or of the body and extremities in sea-water, as in the men and women who take bathers into the ocean; and diseases of the nerves.

Diagnosis.—The important point in the diagnosis of peripheral anæsthesia is the discrimination between it and the anæsthesia, due to central causes. The elements of the diagnosis have been dwelt upon at some length by Romberg, and perhaps needlessly so, for there can scarcely be a case in which any difficulty in forming a correct opinion can arise except in those cases of anæsthesia in which the fifth pair is involved, and they will presently be more especially considered. As regards the cutaneous nerves, the existence of a peripheral cause, and the non-existence of evidences of cerebral or spinal derangement, will be sufficient indications of the nature of the affection. It could scarcely happen that anæsthesia, the result of central lesions, could exist without other marked symptoms being present, not connected with cases of peripheral origin.

Prognosis.—This depends very much upon the cause, and the ability to remove it. In cases of simple division of a nerve, union may be effected after a time, and the functions restored, but, if any considerable portion of the nerve has been destroyed, the case is hopeless. Even when the cause is removed, as may be accomplished for instance in cases due to pressure, a long period often elapses before complete restoration takes place.

---

[1] For a full account of this subject the reader is referred to two papers on "The Relations of the Nervous System to Diseases of the Skin," by Dr. L. D. Bulkley, in the *Archives of Electrology and Neurology* for November, 1874, and May, 1875, and to "A Memoir on Neuroses of the Skin," read before the New York Neurological Society, by Dr. F. Le Roy Satterlee, and published in the *Psychological Journal* for May, 1875.

The Morbid Anatomy and Pathology call for but few remarks after what has already been said. The lesion, whatever it may be, or the functional disturbance if there be no lesion discoverable, is probably situated in the extreme terminations of the cutaneous branches; for otherwise we should expect to find loss of muscular power more frequently associated with the anæsthesia than is actually the case.

But M. Chapoy[1] has shown that there are exceptions to this rule, and especially in regard to the radial nerve; for, in cases of injury or disease of this nerve causing paralysis of motion in the muscles supplied by it, the tactile sensibility is in the majority of instances preserved. This circumstance is explained by the fact that, as MM. Arloing and Tripier[2] have shown, numerous anastomoses exist between the radial and ulnar nerves.

Treatment.—The most important therapeutic measure consists in the removal of the cause. Unless this can be effected, it is useless to attempt other treatment. If this can be accomplished, electricity is the most efficient agent to be employed toward restoring the irritability to the nerves. Sometimes the primary current is to be preferred, at others the induced. In the latter case the wire brush should be used as one of the electrodes, and the anæsthetic parts be stroked with it at each *séance.*

ANÆSTHESIA OF THE FIFTH PAIR.

Symptoms.—These vary according to the seat of the lesion. If the ophthalmic branch alone be implicated, the anæsthesia is situated in the forehead, the upper eyelid, the conjunctiva, and the lining membrane of the nostril. Irritating substances, therefore, coming in contact with the eye or the pituitary membrane, are not felt, though as regards the latter the sense of smell remains.

If the trouble is limited to the superior maxillary branch, the skin of the upper part of the face and the teeth of the upper jaw are insensible. When the inferior maxillary branch is affected, the temporal region, the skin covering the upper and lower jaw, the under lip, the chin, the lining membrane of the mouth, the anterior third of the tongue, and the teeth of the lower jaw, lose their sensibility; mastication becomes difficult, and the saliva flows from the mouth. In either of these cases the seat of the lesion must be anterior to the Gasserian ganglion. When all the branches of the fifth are involved, and, as a consequence, anæsthesia exists throughout the whole of one side of the face, it is very certain that the ganglion is affected, or that the main trunk of the nerve is itself the seat of the disease. Anæsthesia of the fifth nerve due to lesion of the Gasserian ganglion, or of the main trunk, is very generally accompanied by disorders of nutrition and derangement of the senses of sight, smell, and taste. Fungoid growths on the gums and defective circulation in the face are common in such cases; but ulceration of the cornea and congestion of the conjunctiva do not occur unless the lesion is situated in the Gasserian ganglion, or anterior to it in the ophthalmic branch.

---

[1] "De la paralysie du nerf radial," Thèse de Paris, 1874.

[2] "Recherches sur la sensibilité des téguments et des nerfs de la main," *Archives de physiologie*, 1869, p. 33.

The **Causes** of peripheral anæsthesia of the fifth pair are analogous to those which produce the corresponding affection in the cutaneous nerves, cold being the chief among them. Rendu,[1] in his very complete monograph, expresses the opinion that the majority of the cases of anæsthesia of the fifth pair are due to neuritis.

The **Diagnosis** requires a few special remarks, and these may be stated in the form of Romberg's propositions:

"*a.* The more the anæsthesia is confined to single filaments of the trigeminus, the more peripheral the seat of the cause will be found to be.

"*b.* If the loss of sensation affects a portion of the facial surface, together with the corresponding facial cavity, the disease may be assumed to involve the sensory fibres of the fifth pair before they separate to be distributed to their respective destinations; in other words, a main division must be affected before or after its passage through the cranium.

"*c.* When the entire sensory tract of the fifth nerve has lost its power, and there are at the same time derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerve in its immediate vicinity, is the seat of the disease.

"*d.* If the anæsthesia of the fifth nerve is complicated with disturbed functions of adjoining cerebral nerves, it may be assumed that the cause is seated at the base of the brain."

The **Prognosis**, the **Morbid Anatomy**, the **Pathology**, and the **Treatment**, require no remarks additional to those made when peripheral cutaneous anæsthesia was under consideration, except that, as regards the treatment, if the primary current is employed, care should be taken that the tension be not too high, a point to which reference has already frequently been made.

CHAPTER VIII.

NEURAL HYPERÆSTHESIA (NEURALGIA).

UNDER this head I propose to consider the principal painful affections embraced under the term neuralgia. No designation in medical nomenclature has been more abused than this. Any pains, the origin of which cannot readily be ascertained, and many which are well known to depend upon central lesions, are called neuralgic. I propose, in the present remarks, to include under it those affections only which, so far as can be ascertained, are not due to disease either of the brain or spinal cord, but the seat of which is in the nerves themselves. Following the classification of Valleix, I shall consider—

---

[1] "Des anesthésies spontanées," Paris, 1875, p. 107.

a. Neuralgia of the fifth pair.
b. Cervico-occipital neuralgia.
c. Cervico-brachial neuralgia.
d. Dorso-intercostal neuralgia.
e. Lumbo-abdominal neuralgia.
f. Crural neuralgia.
g. Sciatic neuralgia.

NEURALGIA OF THE FIFTH PAIR OF NERVES.

Symptoms.—Either division of the fifth pair of nerves may be the seat of the disease, or all may be simultaneously affected.

1. *Ophthalmic Division.*—This branch of the fifth is distributed to the side of the nose, the eyelids, the lachrymal gland, the globe of the eye, the conjunctiva, the forehead, and the scalp. The long root of the ciliary ganglion communicates with the nerve, and anastomoses take place with the superior maxillary branch.

Valleix has shown that there are particular spots in which neuralgic pains are always more severe than in others, and that these are the points where the nerve either passes through a foramen in a bone, or penetrates a fascia. In the ophthalmic nerve several of these points are to be found. The most prominent is in the nerve as it passes out of the supra-orbital foramen to ramify on the forehead and scalp; another is seated in the upper eyelid; another in the long nasal branch as it passes to the skin through the line of union of the nasal bone with the cartilage; another is located in the eyeball, and another at the inner angle of the orbit. Besides these which are peculiar to the ophthalmic branch, there is another situated near the parietal eminence, and which corresponds to the inosculation of various branches.

The most common form of neuralgia affecting the ophthalmic division of the fifth nerve is hemicrania. The occurrence of the paroxysms is marked by a tendency to periodicity. The pain is exceedingly sharp and lancinating, and occupies the frontal, temporal, or parietal regions, being especially intense at the point corresponding to the supra-orbital foramen, or at least that situated near the parietal eminence. It frequently happens that this latter spot is the only part affected. The paroxysm usually comes on in the morning, and rarely lasts longer than twenty-four hours; frequently it disappears at nightfall. The pain is greatly aggravated by mental or physical exertion, by loud noises or bright lights. It is often complicated with nausea and vomiting, in which case it constitutes what is known as sick-headache. In other cases the pain is mainly confined to the eyeball and the accessory parts. There is then lachrymation, from the fact that the lachrymal gland is supplied from the ophthalmic division, and there may be visual troubles from the relation which the nerve bears to the ciliary ganglion.

This form may also be distinctly periodical in its occurrence, and it rarely lasts longer at one time than twenty-four hours.

2. *Superior Maxillary Division.*—The distribution of this branch is to the teeth of the upper jaw, the lower eyelid, the side of the nose, the upper lip, to the lining membrane of the nose and mouth, and to the temple and cheek. It inosculates freely with the ophthalmic division, and is in intimate relations with the spheno-palatine ganglion.

The painful points of Valleix for this nerve are, in the infra-orbital nerve as it emerges from the infra-orbital foramen to be distributed to the lower eyelid, the side of the nose, and the upper lip; over the most prominent part of the malar bone, where the nerve is very superficial; an uncertain point on the gums of the upper jaw; a similar point on the upper lip, and another on the palate. Neuralgia of this division occurs in paroxysms, and may, like that of the ophthalmic, be periodical in its attacks.

3. *Inferior Maxillary Division.*—This nerve is distributed to the cheek, the tongue, the lower jaw and teeth, and to the sub-maxillary gland. It is also in connection with the otic and sub-maxillary ganglia.

Its painful points are a spot on the auriculo-temporal branch, just in front of the ear; another on the inferior dental nerve, where it emerges from the inferior dental canal, through the mental foramen.

It is generally the case that facial neuralgia is limited to one side, but both are sometimes affected. It may also be confined to very restricted boundaries, the extreme terminal branches alone being involved.

Causes.—According to my experience, facial neuralgia is rarely met with in young persons, but is more common during adult life. It is certainly more apt to attack females than males, and is often transmitted by hereditary influence.

The most common exciting cause is, in this country, malaria, and this is especially the case with the affection in the ophthalmic division, as manifested in hemicrania and supra-orbital neuralgia. This latter is often popularly known as "brow-ague."

Among other causes are to be mentioned mental excitement, anxiety, intense intellectual exertion, exposure to cold and damp, the loss of blood, as in the case of women after childbirth, or from menorrhagia, prolonged lactation, and the changes due to the cessation of the menses.

Another very common cause is syphilis, and there is reason to think that the gouty diathesis may also excite it.

But, as Anstie[1] remarks, it is after the powers of life begin to decline that the most formidable varieties of facial neuralgia are encountered. Those forms which are attended with muscular spasm, constituting the "tic douloureux" of the French, and another still more violent which Trousseau has designated "tic épileptiform," are almost peculiar to advanced life. The pain in these affections is atrocious, and is excited by the least muscular action in the face, by a touch, however light, or even by a breath of air. They are often accompanied by a hereditary tendency to insanity, and they eventually wear away the life of the miserable sufferer.

Facial neuralgia may also result from tumors compressing the nerves, from thickening of the bones, or of the periosteum, causing narrowing of the foramina through which they pass, and from interstitial organic changes taking place in the nerve-trunks.

The **Diagnosis** requires no special remarks, and the **Prognosis** depends upon the cause, and the ability to remove it. In general terms it may be stated that the malarial and syphilitic forms are usually readily cured, while others are seldom thoroughly relieved. The intense varieties, coming on for the first time late in life, are absolutely incurable, and are very seldom capable of even being mitigated.

CERVICO-OCCIPITAL NEURALGIA.

In this affection the pain is situated in the sensory branches of the first four cervical nerves, though the great occipital which arises from the second cervical is mainly the one affected. These nerves are distributed to the occipital and posterior parietal regions, as well as to the neck and lower part of the cheek. The painful points are those at which the nerves become most superficial.

The pain in cervico-occipital neuralgia, though severe, is not in general so intense as that of the facial variety. There is a tendency in the affection to extend so as to involve the inferior maxillary nerve, and, when the disease has lasted some time, a paroxysm rarely occurs without this nerve being implicated. After the acute stage of a paroxysm has passed off, there remains a dull, heavy pain, which continues several days, and which is increased by the pressure of the clothing, by mental exertion, or by moving the head.

The **Causes** are similar in general character to those of facial neuralgia, though cold is probably a still more powerful factor in the etiology.

The **Diagnosis** and **Prognosis** call for no special remarks.

---

[1] Article "Neuralgia," in Reynolds's "System of Medicine," vol. ii., p 726.

CERVICO-BRACHIAL NEURALGIA.

In this form the brachial plexus, the nerves which go to form it—the five lower cervical and first dorsal—and those which arise from it, are the seat of the affection. The pain may therefore be felt in the subclavicular region, along the whole length of the upper extremity, or in the situation of the mammary gland. The exact seat varies of course with the particular nerve affected. It is often accompanied by various sensations of numbness, and interferes more or less with the movements of the limb. The principal painful points are the axillary in the armpit, and corresponding to the brachial plexus, the scapular near the inferior angle of the scapula, the acromial in the angle between this process and the clavicle, the median cephalic in the bend of the elbow, the ulnar corresponding to the most superficial portion of the ulnar nerve at the back of the elbow-joint, and the radial at the point where the radial nerve becomes superficial at the lower part of the forearm.

Among the **Causes** of cervico-brachial neuralgia, rheumatism, malaria, excessive muscular exertion, and injuries, are preëminent. It is not so frequently the result of malaria as the corresponding affection of the facial nerve.

There is nothing special to be said relative to the **Diagnosis** and **Prognosis.**

DORSO-INTERCOSTAL NEURALGIA.

In this affection the dorsal and intercostal nerves are the seat of the pain. In the first case the disease is often regarded as rheumatic or muscular, and has received the popular name of lumbago; in the latter it is often known as pleurodynia. Whether in the dorsal or intercostal form, the pain does not often occur in well-marked paroxysms, but is more or less continuous in character, and is much increased by muscular exertion. In the dorsal form, the mere act of straightening the back causes great suffering, and, in the intercostal, respiration is exceedingly painful.

The painful points are very numerous, and in general correspond to the situations where the nerves become most superficial.

The association of intercostal neuralgia with herpes zoster of unilateral form is an interesting fact, and one which led to the recognition of other skin-diseases as being essentially nervous affections.

The **Causes** of dorso-intercostal neuralgia are cold, rheumatism, malaria, exhaustion, and, in women, the depression of vital power, due to profuse menstruation or prolonged lactation. Anæmia, both in males and females, is also a common cause, however produced.

The **Diagnosis** of the dorsal form is not a matter of difficulty; the intercostal has, however, often been mistaken for pleurisy. The **Prognosis** is more favorable than in the other neuralgias described.

Lumbo-abdominal and *crural neuralgias* are not very common. The latter is seldom a primary affection.

SCIATIC NEURALGIA.

This form is characterized by the occurrence of pain in the course of the sciatic nerve and its branches, mainly in those distributed to the skin. It may be restricted to the gluteal region and upper part of the thigh, or may extend to the sole of the foot or toes. The principal painful points are those which correspond to the sacral foramina, where the large and small sciatic nerves emerge from the pelvis ; a series corresponding to the emergence of cutaneous branches through the fascia, a fibular point at the head of the fibula, an external malleolar, and an internal malleolar.

Sciatica generally begins as a dull, heavy ache, which gradually becomes more and more intense, and which, like all the other forms of neuralgia, is aggravated by muscular exertion. It is subject to exacerbations of violence, during which the least agitation of the body still further increases the intensity of the suffering. Sometimes the pain darts through the nerves like electric shocks, while at others it retains its original situation. It is often accompanied by muscular contractions. Anæsthesia is generally present in the parts which are or have been the seats of the pain, and can readily be detected with the æsthesiometer.

A patient who has once had an attack of sciatica becomes thereby more liable to others. The nerve, after the full force of the disease is spent, remains in a more or less irritable state, during which it is particularly liable to a fresh outbreak, and, even when this does not occur, it is quite common for the patient to be reminded, on any little extra excitation or exposure to cold, that he has a master ready on the least sign of rebellion to put the screws to his refractory subject. These remarks are applicable to all neuralgias, but they appear to me to be specially so to sciatica. Sometimes, even when the individual remains perfectly still and has committed no indiscretion, there are sharp, shooting pains, which follow the course of the sciatic nerve and its branches.

The affection generally lasts two or three months, and is liable to recur.

Causes.—The etiology of sciatica is not materially different from that of other neuralgias, except so far as it is modified by local circumstances. Among these latter, are enlargement of the prostate gland, by which pressure is exerted on the nerve, various tumors of the abdominal organs, the pressure of the fœtal head in childbirth, accumulations of fæces in the large intestine, etc. It is also occasionally induced by the pressure on the nerve which results from sitting long on a hard chair. Several cases of this kind have come under my observation.

I have also noticed the fact that sciatica is often developed with great suddenness on the patient making some unusual exertion of the limb. In such cases the effort is probably only the spark which lights up the flame.

The **Diagnosis** is not a matter of any difficulty, though I have many times seen cases mistaken for diseases of the spinal cord, and *vice versa.* The **Prognosis** depends greatly on the ability to remove the cause.

Morbid Anatomy and Pathology.—The remarks which might be made under this head have already been expressed to some extent in the foregoing pages, and there is not much more that could be said without entering the domain of pure speculation. I may, however, state my opinion that neuralgia, not directly the result of some physical cause interfering with the integrity of the nerve in which it is situated, is almost invariably induced by a depressed state of the system. Its existence in such cases is evidence, therefore, of deficient physical stamina, and of the fact that the nervous system is not duly nourished. The remote factor may be malaria, syphilis, rheumatism, gout, or some other cause capable of lowering the vitality of the organism, and, as a consequence, that of the nerves. It is of course of the utmost importance with reference to the treatment, to ascertain whether there is, or is not, any such constitutional taint, but, whatever the result of our inquiries in this direction, that system of therapeutics is best which, in addition to special medication, embraces restorative means.

Treatment.—The measures which it is proper to employ in neuralgia may be divided into two categories, the constitutional and the local.

Among the constitutional remedies must be included those which are for the correction of any taint which may be present. If there is reason to suspect the existence of syphilis, iodide of potassium is an indispensable remedy, and should be given in large doses. It is also advisable in rheumatic neuralgias, especially of the cervico-occipital region. If malaria can be ascertained to have exerted an influence, quinine must be administered; and, indeed, it is safe to act upon the theory that this has been the cause, unless some other can be clearly made out. It must be recollected that malaria may give rise to neuralgia, especially in the facial nerve, without there having been any other manifestation of its toxic effect; and that the affection is often cured by large doses of quinine, when the patient has not apparently been subjected to the malarious influence. Should there be no relief after three or four ten-grain doses of quinine, it should still not be decided that the disease is not of malarious origin, but the quinine should be given in still larger doses, as in Dr. Clinton Wagner's own case, in which fifty grains were taken in eight or ten hours.[1] If there is still no improvement, arsenic should be administered. I have seen many cases of supraorbital neuralgia, undoubtedly the result of miasmatic poisoning, effectually cured by arsenic, when quinine had failed. From my own experience, I am very well convinced that it acts much more efficaciously when administered by hypodermic injection than by the stomach. Four drops of Fowler's solution, diluted with an equal quantity of water, should be given twice a day, and the quantity should be gradually increased to eight or ten drops at a dose. Even in cases not malarious, arsenic will often be found to be a most valuable therapeutic agent.

If a gouty diathesis is present, colchicum should be used; and, if rheumatism be clearly made out, the blood should at once be rendered alkaline by liquor potassæ.

Whether any specific trouble be discovered or not, general tonics are always indicated; among them cod-liver oil occupies the front rank, and iron is not far behind it in value; strychnia is also very generally useful. Among constitutional remedies, ergot has proved of very decided benefit in my hands, and this especially in sciatica. It should be given in large doses, a drachm or more of the tincture three times a day.

A full and nutritious diet is of great value in the treatment of neuralgia, as are likewise sunlight, and pure and fresh air.

In addition to these purely constitutional measures, there are others which, though administered to act upon the system at large, are given for the purpose of arresting a paroxysm, or deadening sensibility, so as to prevent the pain being felt. The medicines embraced in this category are included among the stimulants, narcotics, and anæsthetics. Opium and its various preparations are preëminent as palliatives of the neuralgic paroxysm, and morphia stands first among them. It is most efficaciously administered hypodermically, in doses varying from one-sixth grain to half a grain, or even more in extreme cases. Great care should be exercised in its use, and the smaller quantity mentioned should not be exceeded except by regular gradations. It is immaterial in what part of the body the injection is made, so far as its influence over the pain is concerned.

But in sciatica it appears to me to be advantageous to follow a plan of treatment which the size of the affected nerve renders admissible, and that is, the injection of morphia into the tissue of the nerve, or, if that should not be effected, as near to it as possible. The preparation I use is a solution of the sulphate of morphia, consisting of ten grains of the salt to two drachms of water as proposed by Dr. Lawson.[1] This mixture is partially solid at ordinary temperatures, and must be warmed before administration, so as to cause complete solution of the separated crystals. Every six minims of this solution contain one-half of a grain of the sulphate of morphia. Two minims, equivalent to the sixth of a grain, are sufficient for a first injection, and this may be gradually increased, if necessary, to half a grain. One injection a day is generally sufficient, but two may be required in extreme cases. Dr. Lawson carries the point of the syringe to the depth of an inch or an inch and a half, without regard to the nerve further than that he recommends the injection be given in its neighborhood. I think it is better, if possible, to strike the nerve with the point of the syringe, and then to inject. To do this is no difficult matter. Select a point on the posterior aspect of the thigh, about four inches below the trochanter major, and an inch exterior to the median line, push the point of the syringe perpendicularly, and at a depth varying from one to two inches the nerve will generally be reached, as will be evidenced by the patient feeling a slight thrilling sensation in the parts below. Then the requisite quantity is to be injected, and the syringe withdrawn. If the nerve should not be touched, the injection may, nevertheless, be made.

---

[1] "Proceedings of the New York Neurological Society," *Psychological Journal*, August, 1874, p. 126.

The effects of this treatment—especially if the injection be made into the nerve—are generally very decided at once. I have had patients, who walked with the utmost difficulty, leave my consulting-room with an elastic step and free from pain a few minutes after such an injection has been made; and others, who could not even turn in bed or move the affected limb, get up and walk about the room after a like period. The pain, however, usually returns, and the injection must be repeated, and in slightly larger doses.

But the treatment is not only palliative; it is often positively curative. Dr. Lawson himself was permanently cured by it, and many cases within my own experience have been equally successfully treated.

In other neuralgias, notably in the cervico-brachial—brachialgia—and the dorso-intercostal—lumbago, and pleurodynia—it is also remarkably efficacious.

Among other medicines of this class are belladonna, or its alkaloid atropia, Indian hemp, aconite, gelseminum, bromide of potassium, hydrate of chloral, hyoscyamus, conium, and some others of minor importance.

Of these, aconite is preferably employed in the form of Duquesnel's aconitia, and is often remarkably efficacious when all other means have failed. It should be used with great caution, and the doses be very gradually increased, till some decided evidence—numbness in the arms and legs, for instance—of its physiological action or the cessation of the pain be obtained. I usually give it in solution in dilute alcohol in the proportion of one grain to the ounce. Four minims of this, equal to the $\frac{1}{120}$ of a grain, are enough to begin with. In two hours a dose of five minims may be given, and so on, if necessary, up to ten minims, or $\frac{1}{48}$ of a grain. Beyond this I have never ventured, and only once have I carried the dose to this point. The patient, a master brass-finisher, had suffered with intense left facial neuralgia, which had resisted every means that had been tried. When he first consulted me, I found some reason to suspect the existence of a syphilitic taint, and I treated him with large doses of iodide of potassium. This was in the early part of August, 1880. He gradually improved, and, while taking one hundred and eighty grains a day, declared himself free from all pain. I continued the remedy for a couple of weeks longer, and then stopped its administration. But in a month's time he returned, with the pain as bad as ever. I again tried the iodide, with mercury in addition, but without the least effect, so far as mitigating his suffering was concerned. I then gave Duquesnel's aconitia in the way mentioned. At eight drops he began to experience relief, though there was a general burning sensation over the whole body, and great physical and mental prostration. I gave nine drops, and two hours afterward ten, with the effect of entirely stopping the pain; and up to this time, four months having elapsed, there has been no relapse. Sometimes it fails. It did so notably in one of the worst cases of spasmodic facial neuralgia I ever saw, and which I brought before the American Neurological Association at its meeting in June, 1880; but I have had several cases of a very severe type in which its effect was all that could be desired, and others in which it produced marked alleviation. Dr. R. F. Weir[1] has reported an interesting case of cure by its use. It is especially useful in facial neuralgia. Gelseminum is also a drug of undoubted power over neuralgia. Dowse[2] expresses the opinion that its action is more specifically exercised upon the dental branches of the inferior maxillary nerve, and I am inclined to concur with him in this view.

---

[1] "Sciatica, Lumbago, and Brachialgia," London, 1872, p. 93. Dr. Lawson uses the muriate of morphia, but, the sulphate being the compound generally employed in this country, I have made the change mentioned.

Of very great value are chloroform and ether, administered by inhalation, and the various forms of alcoholic liquors. It not unfrequently happens that an attack of neuralgia can be at once aborted by an ounce or two of whiskey or brandy, especially in a person not habituated to their use.

A somewhat different class of remedies for neuralgia are those which are either tonic to the nervous or general system, without, as quinine and arsenic, being antagonistic to malaria. Among these are strychnia, phosphorus, and iron.

---

[1] *Archives of Medicine*, August, 1879.

[2] "Neuralgia; its Nature and Curative Treatment," London and New York, 1880, p. 33.

Of these strychnia is, I think, most efficacious when administered hypodermically in doses of from the one forty-eighth grain to the one thirty-second grain twice daily ; or it may be given internally in somewhat larger doses three times a day.

I have long used phosphorus in the treatment of neuralgia. I at one time extensively employed the phosphoretted oil, but more recently have substituted the phosphide of zinc, which, I think, is altogether a preferable form of administration. The formula given on page 69 will fulfill every indication for phosphorus and strychnia. These two remedies are particularly beneficial in neuralgia occurring in persons who have exhausted the vital powers by dissipation and excesses of various kinds.

Iron is especially valuable in cases of neuralgia due to, or accompanied with, an anæmic condition of the system. Anstie speaks highly of the tincture of the chloride, and ascribes to it a marked and direct influence upon the nervous centres different from that produced by other preparations of the mineral. It should be given in doses of thirty or forty minims, properly diluted in water, three times a day. While recognizing the benefit to be derived from this agent, I have generally preferred the sesquioxide in powder, which can be taken without injury to the teeth or probability of stomach derangement. Large doses—twenty to forty grains three times a day—should be employed.

The chief local means of treatment consist of counter-irritation and the application of certain substances calculated to deaden the sensibility of the nerves. Under the former head come blisters and the various stimulating or irritating liniments, essential oils, solutions, and the actual cautery. Blisters should be applied along the course of the affected nerve. They are especially valuable in sciatica. Liniments are rarely of much service, and, moreover, they are dirty. Of the essential oils, that of a species of peppermint put up by the Japanese is sometimes of immediate service in supra-orbital neuralgia, as is also strong aqua ammoniæ. The actual cautery often affords prompt relief, either when applied over the nerve or to some distant part of the body. In sciatica, Erb[1] speaks of the palliative effect of the red-hot iron applied to the external ear. As he very properly remarks, the fact that we cannot explain the *rationale* of its action is no reason why we should ridicule its use.

Of sedative applications, the tincture of aconite, belladonna, opium, etc., are sometimes of service. Dr. Dowse, in the excellent little work already cited, speaks highly of a solution of chloral hydrate applied externally as being efficacious even when the same drug given internally has failed to give relief. His method of using it is to make a solution in the proportion of one ounce of chloral to sixteen of water.

[1] Ziemssen's "Handbuch," Band xii., p. 162.

This is then made hot. Three layers of lint dipped in this solution are next applied to the skin over the affected part, and over these three or four folds of flannel, previously soaked in very hot water and wrung as dry as possible. Over the whole a piece of India-rubber sheeting is placed. The whole is then bound firmly to the surface, and left in position for six or eight hours. When removed, the skin should be painted with collodion or dusted with starch, and then covered with cotton-wool.

I have tried this process in one very severe case of crural neuralgia, and in one of cervico-occipital neuralgia, both of which were obstinate cases, with very gratifying success.

Heat, either dry or moist, is of itself of service in most cases, and cold, in the form of ice, sometimes relieves neuralgic pain very promptly.

Dr. W. H. Thomson,[1] in a recent clinical lecture, ascribes very beneficial results to the local application of water of from 85° to 95° to the skin in cases of sciatica. He considers it especially useful in those instances in which the muscles are contracted.

But, above all local means, not only for relieving the pain of any particular paroxysm, but also for effecting a permanent cure, electricity stands first. I have employed it in every possible form, and am satisfied that the primary galvanic current is the preferable agent. Indeed, I very much doubt if the induced current, unless in a few cases, when the wire-brush has been employed, has ever, in my experience, accomplished any very decided benefit. In the employment of the primary current, the positive pole should be applied over the seat of the pain, and not more than fifteen or twenty Smee's cells should be used. The application should be continuous for at least half an hour, and should be repeated every day for several weeks, and in extreme cases longer. I have cured a number of severe cases of nearly every kind of neuralgia by the aid of electricity when other means had entirely failed. I rarely, however, employ it without at the same time insisting on such constitutional treatment as the case seems to require.

I have several times used acupuncture with success, and have likewise employed electro-puncture with decided benefit in ten cases of sciatica. In either operation the needles should be introduced at the most painful parts, and, when galvanism is also used, the current should be passed continuously through the needles. In a notable case of sciatica which I saw in consultation with my friend Dr. John Gallaher, of this city, a severe attack was at once cut short by electro-puncture. Two or three cells will afford a current of sufficient tension.

As to surgical operations on the affected nerves, either of section or excision of a portion of their continuity, the success which has hitherto followed them has not been such as, in my opinion, to warrant their repetition.

---

[1] "On the Treatment of Sciatica," "American Clinical Lectures," vol. i., No. 6, 1875.

But there are two other surgical means of treatment in certain neuralgic affections which have been recently introduced, and which are entitled to something more than a mere passing reference. These are "nerve-stretching" and "nerve-compression."

The former has been practised mainly on the sciatic nerve, for the relief of sciatica—though it is applicable to other nerves. I have operated on the sciatic nerve in this way five times, and with the result of affording immediate and, up to the present time, complete relief. I have always performed the operation on the nerve at about the junction of the middle and lower thirds, that being the point at which it is more readily reached. I make an incision four or five inches in length through the skin and aponeurosis, and expose the nerve. I then pass under it an ivory paper-knife, and gradually lift the nerve from the bottom of the wound, stretching it to the extent of three or four inches, while making the traction as far as possible in a downward direction. In the last case, I put my index-finger under the nerve, and lifted it out of the thigh with much more ease and with less risk of injury than before, and this is certainly the preferable procedure.

The operation is by no means a painful one; in fact, there is no pain except that caused by the preliminary incision through the skin. I have in two of the cases omitted to use anæsthetics; but in the others I employed the ether-spray, so as to abolish the sensibility of the skin. I prefer that the patient should be sensitive to pain while the stretching is being performed, as important information is derived from the sensations, the object being to carry the extension to the point of producing very decided numbness.

I have never had any untoward accident follow the operation. For several days subsequently there have been numbness and paresis; but these phenomena have gradually disappeared, and without being followed by a return of the pain.

Compression, as a remedy for neuralgia, has been practised to a limited extent, but is, I think, worthy of more extended use. Dowse[1] speaks of it as a "palliative mode of treatment, which certainly has some advantages;" but I have in several cases carried it to a much greater extent than it has hitherto been employed.

My first cases were two of neuralgia of the testis,[2] and in these I subjected the spermatic cord to pressure strong enough to break up the axis cylinder of the spermatic nerve, with the result in both instances of obtaining complete relief from the most agonizing suffering.

[1] *Op. cit.*, p. 27.

[2] "Neuralgia of the Testis," *St. Louis Courier of Medicine*, May, 1880, and *Neurological Contributions*, No. III., 1881.

In neither case has there been any return of the disease nor loss of genital power. I made use of an apparatus similar to a lemon-squeezer, the blades of which could be brought closely together by means of a screw, though in the first case a wooden test-tube-holder answered the purpose. Since the publication of these cases, I have employed compression in two cases of sciatica, using a tourniquet and an ivory ball for the purpose, but with only partial success, and, in one case of severe supra-orbital neuralgia, with complete relief. The cases in which compression is applicable are limited, of course, to those nerves which, by their situation, allow of strong force being exerted upon them, from the fact that they pass over bone, and to those which, like the spermatic nerve, admit of the whole tissue being compressed between two opposing hard substances.

CHAPTER IX.

SYPHILIS OF THE PERIPHERAL NERVOUS SYSTEM.

The peripheral nerves are often the seat of various syphilitic lesions. They may be compressed even to the extent of complete destruction by a syphilitic neoplasm developed in their tract. This compression may occasion a local neuritis with consecutive atrophic degeneration, and it is especially apt to be met with, in connection with the cranial nerves, at those points where they penetrate the dura mater, thickened by a syphilitic infiltration. Sometimes this leads to a slight compression of the nerve, the sheath of which is thickened and the nerve itself softened ; and, again, to anæmia and subsequent atrophy.

It may also happen that a gumma, developed in the vicinity of a nerve, reaches this last by direct growth. This is, above all, liable to occur with those nerves the sheath of which is not very thick, and particularly for the chiasma of the optic nerve. Virchow, Von Graefe, and Heubner, have published cases of the kind.

The peripheral nerves may also be the seat of primitive syphilitic lesions, and this to a considerable extent. They then lose their rounded form and white color, and present the appearance of reddish cords formed of connective or fibroid tissue. At other times they have been found of a lardaceous consistence, or rather swollen and infiltrated by a reddish or yellowish-gray substance. These alterations have, up to the present time, been exclusively observed in the cranial nerves.

Finally, in the instances of certain neuralgic patients, peripheral paralyses have been observed which, persisting until the death of the individual, have presented, on the most thorough examination, no alteration in the corresponding nerves. Syphilitic lesions limited to a single nerve are always manifested by grave functional troubles of the organs supplied by the affected nerve, with integrity of neighboring organs. Generally these lesions concern the oculo-motor nerve. Ptosis is then observed before the movements of the globe of the eye are affected. Later are developed external strabismus, exophthalmia, and a considerable dilatation of the pupil.

When the syphilitic lesion affects the branches of the facial, the ordinary symptoms of paralysis of this nerve are observed. When it is limited to the abducens, there are internal strabismus and diplopia. Paralysis of the muscles of mastication of one side has been noticed, consecutive to a syphilitic alteration of the motor branch of the fifth nerve of the corresponding side.

Peripheral motor paralyses of syphilitic origin are accompanied with abolition of the electric excitability of the branches of the affected nerves (Ziemssen), and with atrophy of the corresponding muscles (Heubner).

Syphilitic lesions may concern the sensory portion of the trifacial, and then a trifacial neuralgia is developed, the origin of which is revealed by its nocturnal exacerbations. Hyperæsthesia sooner or later is replaced by anæsthesia.

Cases of amblyopia and of amaurosis, yielding to the action of specific medication, and evidently due to syphilitic lesions of the optic nerve, have been published. These lesions may also be manifested by hemiopia.

And here and there in medical literature cases are found recorded of neuralgia affecting various parts of the body, and which have been promptly cured by anti-syphilitic treatment.]

Several cases of syphilitic anæmia have come under my observation, in which there was no reason for suspecting a central lesion, and which were promptly cured by the specific treatment recommended in other parts of this work. Cases of syphilitic deafness, due to alterations of the auditory nerve of one or both sides, are by no means infrequently met with, and, unless taken very early in their development, by the most decided anti-syphilitic treatment, are apt to prove intractable.

Aphonia, due to paralysis of the laryngeal muscles, from syphilitic alterations of the communicating branch of the spinal accessory nerve or the recurrent laryngeal nerves, is not a very uncommon occurrence, and is usually, if subjected to treatment before the excitability of the muscles is lost, a not obstinate affection.

In all cases of syphilitic paralysis, in addition to the specific treatment imperatively demanded, electricity should always, if possible, be employed. I mention this last fact particularly, because I am led to believe the idea is more or less prevalent, that such paralyses require no other treatment than iodide of potassium and mercury. I have repeatedly found in cases of facial paralysis, clearly syphilitic in character, that the electric contractility of the muscles to the faradaic current was entirely abolished, and that the galvanic current of strong tension was necessary to excite them to action.

SECTION V.

DISEASES OF THE SYMPATHETIC NERVOUS SYSTEM.[1]

CHAPTER I.

PATHOLOGY OF THE CERVICAL SYMPATHETIC.

WHATEVER idea we may form of the relations of the great sympathetic with the cerebro-spinal axis, and of the nature of the nervous functions which devolve upon it, it is incontestable that the nerve exercises an immediate influence over the circulation, calorification, the secretions, and the nutrition of the organs to which it is distributed, and over the elements comprised in these organs. The well-known experiment of Claude Bernard, which consists in dividing the great cervical sympathetic, is quite adequate to exhibit these several influences. As a consequence of such a section there result :

1. A very apparent dilatation of the vessels of the face and of the ear of the corresponding side, and which amounts to a well-marked congestion of these parts. Nothnagel has been able to convince himself, by direct observation, that these vascular troubles affect also the membranes and the encephalon of the corresponding side.

This dilatation and congestion has been attributed to a paralysis of those vaso-motor fibres of the sympathetic which give tone to the vessels. If, in fact, a continuous galvanic current be passed through the peripheral segment of the divided nerve, we see the vessels resume their normal calibre, and at the same time the phenomena of congestion disappear.

2. An elevation of local temperature, especially manifested in the natural cavities (auditory canal, nostril, mouth), on the side with the

---

[1] Although there is, in my opinion, scarcely sufficient data relative to the diseases of the sympathetic nervous system to warrant the subject being fully considered in a systematic treatise like the present, I have thought it well to incorporate Dr. Labadie-Lagrave's excellent synopsis, which he prepared as an appendix to his French translation of this work.

section. The difference of temperature between one side and the other may reach as much as 1·5° Centigrade.

This local elevation of temperature is in part the result of the vascular congestion determined by the division of the vaso-motor nerves. We know that the temperature of the peripheral organs is, in general, more elevated when an increased quantity of blood circulates through them. But, as this local rise of temperature remains after all trace of vascular congestion has disappeared, it has been supposed that the vaso-motor nerves exercised a direct influence over the function of calorification of the tissues ; that the vaso-motor fibres moderate to a certain extent the nutritive changes of the organs to which they are distributed, and that, moreover, their paralysis causes an increase in local organic combustion, and consequently augmented temperature. This explanation seems to be all the more reasonable, from the fact that, at a later period, the local elevation of temperature is succeeded by a burning which coincides with a local denutrition of the tissues, as, for instance, hemiatrophy of the face. At the same time, in this second period, the paralyzed side does not perspire, or, if it does, perspires less in amount and less frequently than the sound side (Nicati), while during the period immediately after section of the great sympathetic we observe :

3. A decided diaphoresis with epiphora, very exactly limited to the half of the face on the same side as the section.

4. Contraction of the pupil and of the palpebral opening, with retraction of the globe of the eye on the same side as the section. The contraction of the pupil, consequent on section of the great cervical sympathetic, is easily explained if we admit that this nerve has control of the radiating fibres of the iris. When these fibres are paralyzed, the irian sphincter, no longer having its action resisted, contracts, and the pupil is diminished in size. The narrowing of the palpebral opening and the retraction of the eyeball have been attributed to paralysis of the orbital muscle of Müller innervated by the sympathetic, and which has for its function the counteraction of the straight and oblique muscles of the eye, and of preventing these muscles from drawing the globe backward.

In exciting the cervical sympathetic with the galvanic current, for example, we observe a certain number of phenomena, the reverse of those which result from its section. The pupil is dilated, the eyelids are widely opened, and the eyeball is projected forward ; the vessels are contracted and the circulation restricted, at the same time that the temperature is diminished in the corresponding half of the face. The sensibility is diminished on the same side, the cornea and conjunctiva become dry, and the convulsions caused by strychnia are less pronounced than on the opposite side (Waller, Budge, Claude Bernard, and Brown-Séquard).

These experimental results accord perfectly with those obtained by clinical observation in man. In a certain number of cases of traumatic lesions of the cervical sympathetic, published during the last twenty years, there have been mentioned with variable frequency the different oculo-pupillary and vaso-motor troubles that can be produced by electrizing the sympathetic.

[Fourteen cases of functional troubles of the cervical sympathetic, consecutive to traumatic lesions of this nerve, are then given on the authority of Weir Mitchell, Verneuil, Seeligmuller, Bärwinkel, and others, and eleven cases, from Panas, Poiteau, Eulenburg, and others, of similar derangements from the presence of tumors in the course of the nerve.

Spontaneous paralysis of the cervical sympathetic is also known to be produced, in appearance at least, although not very often. Six cases cited from Bärwinkel, and one from Nicati, are given in illustration, in all of which there were vaso-motor and oculo-pupillary troubles, limited to one side, and in all respects similar to those following section of the nerve.

Besides these, there may be functional troubles of the cervical sympathetic, as consequences of spontaneous or traumatic lesions of the nervous centres. Bärwinkel gives the case of a man who, having the symptoms of bulbar sclerosis, presented also a certain number of phenomena peculiar to paralysis of the cervical sympathetic, and Seeligmuller that of a woman who had like symptoms, in conjunction with cerebral hæmorrhage.—W. A. H.]

In 1869 M. Rendu collected, in a very interesting memoir, a certain number of cases, in which traumatic lesions of the marrow were complicated with functional troubles of the cervical sympathetic, some attributable to paralysis, and others to excitation of the nerve. As in the instances we have cited, oculo-pupillary derangements were most frequently observed. Of eighteen cases referred to by him (fractures, luxations, and wounds with cutting instruments), implicating the cervical sympathetic, the pupil was contracted on the side of the lesion fourteen times. Sometimes there was noted a narrowing of the palpebral opening, with injection of the conjunctiva, the face, and the ear, and elevation of temperature in the same parts. These vaso-motor troubles, complicating contraction of the pupil and of the palpebral opening, were particularly marked in a case cited by M. Rendu from M. Brown-Séquard.[1]

All these facts sufficiently demonstrate that the superior portion of the spinal axis exercises over ocular innervation, and the circulation and the calorification of the cephalic extremity, an influence analogous to that of the great cervical sympathetic. They also go to show that

---

[1] *Archives générales de Médecine*, tome xiv., 1869, p. 286.

the cervical sympathetic draws a great part of its nervous action from the superior segment of the spinal cord.

[A like inference must also be drawn from the fact that in locomotor ataxia there are similar disturbances in the movements of the pupils —disturbances which, as we have already seen, are among the earliest symptoms of the spinal affection.—W. A. H.]

CONCLUSIONS.

From a consideration of the preceding facts, we see that of fourteen cases of traumatic lesion of the region of the neck, in which there were also functional troubles of the cervical sympathetic, ten were of the form of paralysis, while in four only were the symptoms indicative of irritation. In compression of the cervical sympathetic by tumors, of eleven cases, eight were manifested by paralysis, and three only by irritation. And in instances of the compression of the sympathetic by intra-thoracic tumors, functional troubles affected the cervical portion of the nerve, and they were always of a paralytic character.

The troubles consist, for the most part, of oculo-pupillary phenomena—constriction or dilatation of the pupil and of the palpebral opening. More rarely circulatory and calorific troubles (such as congestion with local elevation of temperature in the case of paralysis of the great sympathetic, paleness of the countenance, with lowering of temperature in the case of irritation of the nerve), and derangement of the secretions, are noted. In one case, the compression of the sympathetic by one of the lobes of an hypertrophied thyroid gland caused no other symptom than an increased secretion of sweat.

In a certain number of cases of traumatic lesions of the cervical sympathetic, oculo-pupillary and vaso-motor troubles are complicated with an atrophy of the half of the face on the same side as the lesion. M. Nicati is therefore wrong, in his rather theoretical description of the course of the morbid phenomena in cases of paralysis of the great sympathetic, in considering hemiatrophy as a symptom of the late period of the disorder. In Case III., hemiatrophy was one of the phenomena of irritation of the sympathetic, along with mydriasis, paleness of the corresponding side of the face, and depression of temperature of the external auditory canal; and, in Case VII., hemiatrophy was developed a short time after the accident which caused the paralysis of the sympathetic.

We also notice that, in a case reported by Willebrand (XVII.), the use of iodine preparations caused, not only the disappearance of a subclavicular strumous tumor, but also the paralysis of the cervical sympathetic, which had justly been attributed to the compression of the nerve by the growth. M. Verneuil has seen the dilated pupil of an individual who was the subject of a cervical abscess resume its normal diameter when the abscess was opened and emptied of its contents.

[A like fact has come under my own observation. It occurred in a lady upon whom I had operated for a multilocular cystic tumor of the neck, immediately over the sympathetic nerve. After the excision, a large cavity was left, which was filled with lint for the purpose of arresting the oozing of blood. In a short time symptoms of irritation of the pneumogastric and sympathetic nerves were developed. These consisted of vomiting, irregular respiration, and great disturbance of the heart's action (pneumogastric), and dilatation of the pupil and paleness of the face on the side of the lesion (sympathetic). On removing the pledgets of lint, both series of phenomena at once ceased. —W. A. H.]

CHAPTER II.

NEUROSES OF THE CERVICAL SYMPATHETIC.

MIGRAINE, OR HEMICRANIA.

LATELY certain neuroses have been regarded as functional troubles of the sympathetic nerve. Sometimes they are apparently the result of an irritative action, and, again, of a paralytic state of various parts of this nerve.

By *migraine*, or hemicrania, we understand a painful paroxysmal affection, limited to one half of the head, and which is accompanied by oculo-pupillary, circulatory, and calorific disturbances, which serve to distinguish the painful crises in question from supra-orbital, temporal, or occipital neuralgias, with which they are often, nevertheless, confounded.

The attacks are, in general, of irregular succession, and, in the intervals between them, the patient is apparently entirely well. Usually, the approach of a paroxysm is announced by prodromatic phenomena. The patient is irritable, and indisposed for mental labor. Among other premonitory signs may be mentioned yawning, noises in the ears, the presence of dark specks (scotoma) in the visual field, sneezing, a feeling of constriction in the side of the head to be attacked, and, above all, nausea.

The hemicranial pain generally attains to its highest point in a gradual manner. More frequently it is seated in the left than in the right side of the head, but it may in the same patient attack each side alternately. The pain, instead of being lancinating or boring, as in the case of neuralgia, is rather constricting, and covers a great area. Ordinarily, it is sharpest in the frontal, occipital, or parietal region. There are no painful points, as in neuralgia. Often, however, pressure over a circumscribed part of the parietal region causes an exacerbation of the hypersensitiveness to pain. Ordinarily, also, pressure over the superior or middle cervical ganglion is painful. More rarely, a like effect follows strong pressure made over the spinous processes of the cervical and first dorsal vertebræ. We may state, also, that Dr. Berger has shown with the æsthesiometer that a certain degree of tactile hyperæsthesia exists on the half of the face corresponding with the affected side.

In addition to the pain, we have to call attention to other prominent symptoms which are habitually present. These are nausea and vomiting, hallucinations, such as noises in the ears, circles of fire in the eyesight, and a disagreeable taste in the mouth. Then come, also, oculo-pupillary and vaso-motor troubles, which, according to Eulenburg, may be of two distinct clinical types :

1. Sometimes it is observed that, during the duration of the paroxysm, the pupil on the affected side is manifestly dilated, at the same time that the ball of the eye is retracted to the bottom of the orbital cavity. The corresponding half of the face and the ear of the same side are of an extreme paleness, and the temporal artery is contracted and forms a hard cord, much less prominent than that of the opposite side. The temperature of the external auditory canal is lower than that of the same side—0.4° to 0.6° C. It is also a matter of demonstration that every cause capable of diminishing the flow of blood to the painful half of the head augments the suffering. This is especially shown if the carotid artery of the affected side be compressed. If the opposite carotid be compressed, the pain is lessened.

Toward the end of the paroxysm, when the hemicranial pain is on the point of beginning to disappear, the pupil contracts, the pallor of the face and of the ear of the painful side is replaced by a state of congestion, with sensation of heat, elevation of local temperature, injection of the conjunctiva, epiphora, and acceleration of pulse. The painful crisis very often ends by the appearance of a profuse sweat, an abundant flow of urine, or by a diarrhœal flux. As is readily perceived, the symptoms of this form of hemicrania recall to our minds the results due to traumatic or experimental excitation of the cervical sympathetic. It is for this reason that it has been proposed to give to this clinical form the name spasmodic or sympathico-tonic hemicrania.

2. At other times the oculo-pupillary and vaso-motor disturbances are presented with characteristics absolutely the reverse of those to which we have called attention. During the duration of the paroxysm the pupil is contracted, as is also the palpebral opening, and the upper eyelid droops. The face and ear of the affected side are injected, and the temperature of the external auditory canal exceeds by 0.2° to 0.4° C. that of the unaffected side. The dilated temporal artery beats with force, the pulse is often rendered slower, and compression of the carotid artery of the painful side diminishes the pain. Toward the

end of the paroxysm these symptoms generally change. This form of hemicrania has been called angio-paralytic or neuro-paralytic, for the reason that the symptoms which constitute a paroxysm are exactly like those which are observed on section of the great cervical sympathetic.

Cases have been reported in which the paroxysms have in the same patient alternated in character—the angio-paralytic appearing at one time, and the angio-spastic at another. At other times, the hemicranial pain constitutes the only phenomenon of the accession, the vaso-motor and oculo-pupillary troubles being entirely absent.

As we have already remarked, the paroxysms of angio-spastic and angio-paralytic hemicrania realize with the utmost exactness the morbid picture observed as the consequence of excitation or section of the cervical sympathetic nerve. On this account, certain authors, and particularly Du Bois-Reymond, and Eulenburg, in Germany, have not hesitated to invoke the mechanism in question in the pathogeny of this painful neurosis. Angio-spastic hemicrania should, accordingly, have its point of departure in a periodical irritation of the great sympathetic or of the superior cervical ganglion; while a paralytic condition of these organs is considered to be the cause of angio-paralytic hemicrania.

But this theory has met with opposition. Thus Drs. Brown-Séquard and Althaus have insisted that vascular spasm of one half of the encephalic extremity would naturally produce anæmia of the corresponding cerebral hemisphere, and that such a disturbance of the circulation would cause epileptiform convulsions of the opposite half of the body. But Eulenburg remarks with much reason that electrization of the central extremity of the diseased cervical sympathetic produces a vascular spasm on the corresponding side of the head and of the encephalon, and at the same time the oculo-pupillary and vaso-motor troubles described above. Moreover, the like results are obtained in cases of traumatic irritation of the great cervical sympathetic. Finally, this author asks whether irritation of the nerve in question does not rather induce a partial ischæmia, limited to certain regions of the encephalon, than generalized anæmia extending over the half of this organ.

But if the spasm or relaxation of the vessels of a half of the cephalic extremity, when compared with the like conditions obtained by experimental physiology, enables us to account for the vaso-motor and oculo-pupillary disturbances observed in the course of one or the other form of hemicrania, how are we to explain the principal symptom, pain? Are we to place the seat in the nervous ramifications, which the trigeminus supplies to the dura mater, or in those which the same nerve, as well as the sympathetic, sends to the vascular network of the pia mater? According to Du Bois-Reymond, the pain in the angio-spastic form has no other cause than the tetanic contraction of the non-striated fibres of the vascular walls. It has its analogue in cases of contractions of the muscles of the calf of the leg, of the uterus, and of the intestinal walls, either one of which produces painful sensations. This explanation, which does not lack ingenuity, can only at most be applied to one of the forms of hemicrania.

Eulenburg has proposed, in place of this theory, one of his own, which he thinks is applicable to all cases. In his opinion, hemicranial pain has its point of departure in a disturbance of the circulation, either as anæmia or hyperæmia of the affected cerebral hemisphere. This circulatory trouble acts as a veritable irritant to the sensory nerves of the skin, the scalp, and the meninges, and thus develops the painful paroxysms of migraine.

[By some pathologists, and notably Anstie [1] and Clifford Allbutt,[2] migraine has been regarded as a neuralgic affection of the ophthalmic branch of the fifth nerve, the latter, however, contending for the simultaneous existence of cephalic and abdominal complications. This view must, I think, give way to that which ascribes the main causative influence to derangement of the sympathetic nerve.

But my own experience does not lead me to the extent of accepting the theory of Du Bois-Reymond, that migraine is always the result of a contraction of the vessels—a tetanus, in fact, of the muscular coat; nor to that of Möllendorf,[3] according to which it is always due to a relaxation of the vessels and an increased flow of blood to the brain. I am quite sure, with Eulenburg and Gutman,[4] that there are cases under each of these heads, a view which is also held by Berger.[5] Clinical experience is so decidedly in favor of this latter theory, that it appears impossible to resist the conclusion to which it leads, for we find in practice that those agents which diminish the tone of the arteries cure some cases and aggravate others, while those remedies which increase the arterial tension are sometimes efficacious and again injurious. The importance, therefore, of making an exact diagnosis of the forms of hemicrania with which we have to deal cannot be over-estimated; but, with this end in view, not only should inquiries be instituted relative to the appearance of the face, as regards pallor or redness and temperature, the oculo-pupillary phenomena, and the effects of such remedies as may previously have been given, but ophthalmoscopic examination should be made, if possible, at different times throughout the duration of the paroxysm. Möllendorf observed that, in the eye of the affected side, the fundus was of a bright scarlet color, while in that of the opposite side it remained of its normal brownish-red hue. I have not only frequently noticed this appearance, but in other cases have witnessed a pallor of the fundus of the eye on the affected side, only to be explained on the hypothesis of a diminished amount of blood being in the encephalic arteries on that side. Information of important diagnostic value can also be obtained by observing the effect of pressure on the carotid artery during the period of the seizure. In the anæmic form the procedure causes an aggravation of the suffering, while in the hyperæmic it produces prompt mitigation of the pain and other attendant phenomena.—W. A. H.]

---

[1] "Neuralgia and the Diseases that resemble it," New York, 1872, p. 154.

[2] "On Migraine," *Practitioner*, January, 1873.

[3] "Über Hemicranie," *Virchow's Archiv*, Band xli., p. 385.

[4] "Physiology and Pathology of the Sympathetic System of Nerves," translated by Napier, London, 1879, p. 65.

[5] "Zur Pathogenese der Hemicranie," *Virchow's Archiv*, Band lix, H. 3 and 4, 1874; also, translation by Dr. Gradle, *Chicago Journal of Nervous and Mental Diseases*, July, 1874, p. 296, *et seq.*

Treatment.—The vaso-motor theory of migraine has been in part our guide to the treatment, and the efficacy of the means employed has given us data for successful management, which, though based on empiricism, are of great value.

Thus, it was reasonable to conclude, *a priori*, that the functional trouble of the great sympathetic could be alleviated by the electric current, and experience has established the wisdom of this deduction. J. Benedict, Frommhold, Freber, Rosenthal, and Althaus, as cited by Eulenburg,[1] have published cases of migraine treated with success by galvanization of the great sympathetic. Holst,[2] basing his procedure on the polar theory of Brenner, advises the following method for the galvanization of the cervical sympathetic: In the angio-plastic form of migraine, in which it is necessary to moderate the irritability of the nerve, a current from ten to fifteen elements should be employed, the positive pole being applied over the sympathetic, and the negative held in the hand of the same side. Each *séance* should last for from two to three minutes. In the angio-paralytic form, the negative pole should be applied over the course of the nerve. To obtain a more energetic action on the nerve, the current should be frequently interrupted, or even reversed.

[In point of fact, however, according to my experience, it is a matter of no consequence what the direction of the current is in either form of the disease. One pole should be applied over the nerve in the neck, and the other placed, preferably, on the pit of the stomach, and the action continued for from two to five minutes.

Frommhold,[3] however, advises the use of the faradic current in the affection, and Freber[4] agrees with him in this practice. In my experience, it cannot be compared in efficacy to the galvanic current, and, indeed, I have often found it to aggravate the pain. When it is used, the interruptions should be very rapid, and the intensity as great as the patient can endure.

---

[1] Ziemssen's "Handbuch der speciallen Pathologie und Therapie," Band XII., p. 28; also, "Physiology and Pathology of the Sympathetic System of Nerves," by Eulenburg and Gutman, Napier's translation, London, 1879, p. 70.

[2] "Dorpater medic. Zeitschrift," Band II., 1871, p. 261.

[3] "Die Migraine und ihre Handlung durch Electricität," Pesth, 1868.

[4] "Compendium der Electrotherapie," Wien, 1869.

During the intervals between the attacks, galvanism should still be employed as a remedial agent, with the view of altering, if possible, the tendency to the occurrence of paroxysms; but it is then not the chief therapeutical agent. Indeed, I am not disposed to think that it is ever entitled to this distinction.

In my own practice, during the existence of the paroxysm, I first endeavor to ascertain the character of the seizure. If it is of the angiospastic variety, that is, the form in which the calibre of the blood-vessels is diminished, I administer a large dose of morphia, say the quarter or third of a grain, by hypodermic injection, and at the same time cause the patient to take repeated inhalations of the nitrite of amyl. Latterly, I have sometimes, for the nitrite of amyl by inhalation, substituted the internal administration of this drug in doses of from one to four or five drops, or of glonoine in doses of the one-tenth to half a drop, taken on a lump of sugar.

If there is reason to suspect the influence of malaria in the production of the disease, I give a large dose (from twenty to forty grains) of the sulphate of quinine, instead of the morphia and other substances mentioned. Experiments which I performed, in conjunction with Roosa,[1] show that under the action of this agent the amount of blood in the brain is increased. As these experiments bear directly on the question at issue, I may be permitted to quote them here. It is well known that the obvious phenomena which result from a large dose of quinia are such as indicate an increased flow of blood to the brain. The redness of the face, the injection of the conjunctivæ, the noises in the ears, the sensation of distention or fullness or constriction felt in the head, are all so many indications of cerebral hyperæmia. Still, I was desirous of settling the matter by direct experiment and the employment of those instruments of precision which the progress of science has put at our disposal.

With this object I resolved to take quinine myself, and to have my friend Dr. Roosa, whose abilities as an ophthalmologist and aurist are indisputable, examine the fundus of the eye and the tympanum before the ingestion of the quinine and during the continuance of its effects.

The experiment was made on the evening of May 7th, and I subjoin his report in his own words:

"Vision normal	$= \frac{20}{20}$
Refraction	emmetropic.
Pulse	90

---

[1] "The Influence of the Disulphate of Quinine over the Intra-Cranial Circulation," *Psychological and Medico-Legal Journal*, October, 1874.

"Ocular conjunctivæ white, decidedly free from hyperæmia. Palpebræ congested at outer and inner canthus. Has no tinnitus aurium. Membrana tympani entirely free from evidence of vessels. No congestion along handle of malleus.

"Ophthalmoscopic examination of both eyes reveals a remarkably clear optic papilla on both sides. Arteries and veins, vertical and horizontal vessels, clearly cut, and whole papilla sharply defined, rather paler than congested.

"Took grs. x. sulphate of quinine at 8.30 P. M. At 9 P. M. ocular conjunctiva is congested at outer and inner canthus. Palpebral conjunctiva markedly congested over whole surface. No change in optic papillæ or in drum-heads.

"9.15. Surface of optic papillæ pinkish; arterial vessels seem more distinct; no change in appearance of drum-heads; no tinnitus aurium.

"10. Head feels full; left ear rings; auricles burn; face is decidedly flushed; auricles red, especially lobe of right, where there is a localized congestion that is so marked as to resemble an ecchymosis. There is now a vessel along each malleus; optic papillæ are pinkish. Pulse 84 and fuller.

"10.30. Right drum-head is very much injected along handle of malleus and upper margin. Left less so, but yet injected. Both papillæ very pink, left more so than right. Face flushed, eyes suffused, ocular conjunctiva decidedly congested. Slight headache; tinnitus in both ears.

"11. Redness of auricles diminishing, especially the circumscribed spot on the lobe of left one; face still flushed; tinnitus continues; no headache; feels exhilarated. Drum-heads still injected along malleus; not more so, however, rather less; optic papillæ have a decidedly pinkish hue; no more lateral vessels seen, however; right is especially pink. Tinnitus still continues; vision normal. No further observations were made."

That the phenomena indicated cerebral hyperæmia is self-evident, and therefore no further remarks on the point are necessary.

But it was possible to determine the question with even greater certainty.

I therefore trephined a medium-sized dog, and screwed a cephalohæmometer into the opening in the skull made by the trephine, so that the fluid in the glass tube stood at zero.

I then introduced into the cellular tissue of the abdomen ten grains of sulphate of quinine, dissolved in water acidulated with sulphuric acid. This was done at 3.30 o'clock P. M.

At 3.35 the fluid had risen one degree on the scale (= $\frac{1}{10}$ inch). It continued to rise gradually but steadily, till at 4.10 it had passed over ten degrees (= 1 inch of the tube). At 4.30, one hour after the injection of the quinine, the fluid was at + 15°. It continued at this point till 5.10, when it began to fall, and at 8.15 was at zero. It remained stationary for over an hour, at no time falling to the minus division of the scale.

The stage of excitement scarcely lasted fifteen minutes. It was succeeded by a state of sedation during which the salivation was excessive, and the animal appeared very much as if under the influence of a full dose of alcohol. As the normal condition of the dog was regained, the fluid fell in the tube, and reached the zero almost simultaneously with the disappearance of the symptoms of intoxication.

I repeated the experiment on different days with variable doses of quinine—from two grains to fifteen—in all, four times, and invariably with the result of a steady rise of the fluid in the tube as the effect of the drug increased, and its fall to the zero as the influence wore off. At no time did the fluid reach a lower point than that at which it stood before the administration of the quinine.

I think the several experiments detailed in this memoir show conclusively that the influence of the sulphate of quinine over the intracranial circulation is that of causing hyperæmia and congestion.

So far as I am aware, there are no experiments on record such as I have described, and the theory that the sulphate of quinine produces cerebral anæmia is one not based on fact, but solely on the interpretation of certain phenomena to accord with a previously formed hypothesis of its physiological action.

I think, therefore, that quinia may properly be regarded as an antagonist to the tetanic condition existing in the angio-spastic form of migraine—in addition to its anti-periodic virtue. Experience shows that the effect is almost always an abrupt cutting short of the paroxysm, especially if the nitrite of amyl be inhaled repeatedly, and to the extent of obtaining the full physiological effect of the drug.

In this angio-spastic form of migraine, the treatment in the intervals between the paroxysms should consist of the administration of some one of the bromides (sodium, potassium, calcium, or ammonium) in doses of at least fifteen grains three times a day; for, although the influence of these remedies is to diminish the amount of blood in the brain, they are antagonistic to all forms of muscular spasm. Either one of those mentioned may be advantageously given, in combination with pepsin and charcoal, as recommended under the head of "cerebral congestion." If this method of treatment be followed out for two or three months with firmness and persistency, a cure may reasonably be expected in the great majority of cases.

The treatment during the paroxysm in the angio-paralytic form should be in many respects the very opposite of that proper for the angio-spastic variety. So far, however, as the use of electricity goes, no change is necessary, and a seizure may sometimes be cut short by the galvanic current, from ten to fifteen elements.

Cold to the nape of the neck is also of great value, and compression of the carotid on the affected side is a ready and prompt means of aborting a seizure in many cases. It should be continued in some instances for an hour, or even longer, and then the pressure should be very gradually removed.

For internal medication, a large dose—thirty or forty grains—of guarana or paullinia sometimes acts like a charm, as does also strong coffee, or, better still, caffeine. In some instances strong tea will arrest a paroxysm when coffee has entirely failed.

But it is not wise to rely entirely on any one of the measures. Cold, compression of the carotid, and galvanism, should be employed at the same time, and some one of the internal remedies administered, to be followed by another, if it is not quickly efficacious.

After the paroxysm is over, the real curative treatment should be begun, and this should, as in the other form, consist of the bromides, but in combination with ergot. I usually give the mixture just recommended, substituting the fluid extract of ergot wholly or in part for the water, and continuing the treatment for several months. It is rare that a case resists this form of management.

And, in both varieties, attention should be paid to the hygiene of the patient. The diet should be simple but nutritious. It is an undoubted fact that many attacks of migraine can be directly traced to indulgence in some article of food which the patient knows at the time is almost certain to produce a seizure. When such is the case, an emetic will often prevent the development of more than the premonitory symptom of a paroxysm.—W. A. H.]

CHAPTER III.

PATHOLOGY OF THE THORACIC SYMPATHETIC.

The great thoracic sympathetic nerve controls the vaso-motor innervation of the superior extremities, the trunk, the intra-thoracic organs, and the spinal cord. Physiological experimentation upon the ganglia and nerve of that portion of the great sympathetic leads to the production of phenomena analogous to those which result from the excitation and paralysis of the cervical sympathetic. Still, up to the present time, we have at our disposal only a small number of instances of vaso-motor troubles consecutive to lesions of the thoracic sympathetic, or of the organs to which it is distributed.

We have seen, from the cases previously given, that the compression exercised by aneurisms of the arch of the aorta and of the thoracic portion of this vessel on the left sympathetic reacts ordinarily on the cervical sympathetic of the same side, and that it gives rise to oculo-pupillary and, more rarely, to vaso-motor disturbances of the corresponding side of the face. In addition, the phenomena in question were accompanied in a certain number of cases by an acceleration of pulse, attributed by authors to the compression of the cardiac filets of the great sympathetic, which is regarded as an accelerator nerve of the heart.

Besides these vaso-motor troubles of the face consecutive to compression of the thoracic sympathetic by an aneurism of the aorta, we must note the redness of the cheeks, with elevation of local temperature, observed in cases of pneumonia. M. Gubler, who has specially studied this phenomenon, has called attention to the fact that, in unilateral pneumonia, the redness is almost always limited to the cheek of the corresponding side. On the other hand, it is established from the researches of M. Lepine that, in pulmonary affections, such as pneumonia and tuberculosis confined to one lung, there exists a very notable difference of temperature in the two sides of the body—a difference which is almost always in favor of the side corresponding to that of the diseased lung. This difference may amount to one or two degrees Centigrade at the extremities of the limbs, while in the axillæ it is only a few tenths of a degree.

Quite recently Seeligmuller, in a memoir which we have had several occasions to cite, has called the attention of physicians to the adhesions which, in the case of lesions of the summit, the lung, and its pleural covering, contract with the thoracic sympathetic. This morbid condition occasions an irritation of the nerve, to which a state of paralysis succeeds. In the same way we account for the congestive spots on the cheeks and the dilatation of the pupils observed in tuberculous individuals. In a certain number of cases of unilateral tuberculous lesions, these oculo-pupillary and vaso-motor disturbances are limited to the side corresponding to that of the diseased lung. Thus, in the instance of a woman twenty-four years old affected with catarrh of the apex of the left lung, there was mydriasis of the same side. In a man thirty-eight years of age, who had pleuro-pneumonia of the left side during ten years back, and a large cavity in the apex of the same lung, it was observed, six months before the fatal termination, that at times the cheeks and the ears, markedly on the right side, were subject to intense congestion, while, simultaneously, the pupil of the same side was contracted. In the case of a woman sixty-seven years old, affected with pleuro-pneumonia of the lower lobe of the right lung, there was developed a bed-sore, limited to the buttock of the same side, and which took four months to heal. Six months after the cure of the pleuro-pneumonia, the phenomena of paralysis of the sympathetic—a marked contraction of the pupil and of the palpebral opening of the same side—were observed. Indeed, the left side of the face and corresponding ear were often the subjects of congestion, which made a marked contrast with the paleness of the right side of the face. Again, in a man thirty-one years of age, who, within the period of a year, had suffered from five attacks of pleuro-pneumonia of the right side, the pupil of the corresponding side was contracted. The patient had himself observed that on this same side there was a profuse perspiration whenever he exercised physically, and, above all, when he drank.

Dr. Fleischman,[1] of Vienna, has called attention to like facts, in a work on chronic pneumonia of the apex of the lung in infants. He has shown that in such cases there are vascular troubles limited to one side of the face or head, and transient unilateral erythema, with elevation of the local temperature.

CHAPTER IV.

PATHOLOGY OF THE ABDOMINAL SYMPATHETIC.

THE great sympathetic contributes to form, with the vagus, the several plexuses which preside over the function of innervation of the organs contained in the abdominal cavity. The disorders which have their seat in these plexuses are manifested by painful sensations, by motor troubles, by exaggerated or insufficient movements of the contractile tissues which enter into the constitution of the abdominal organs, and by circulatory and secretory troubles.

In the affection known as cramp of the stomach (gastralgia), there is a veritable contraction of the walls of this organ, which produces compression of the terminal extremities of the nerves of the stomachal plexus and violent pain. The pain is, hence, only a consequence of the motor trouble. However, for M. Sée the gastric pain is generally due, not to a contraction, but to a distention of the stomach by gas, and the consequent stretching of the nerves resulting therefrom.

The centres which regulate the secretory function of the stomach appear to be situated in the walls of this organ. In fact, the division of the pneumogastric nerves, as well as the destruction of the plexus of Auerbach and of Meissner with ammonia (Schiff), has no influence over digestion, and consequently over the secretion of gastric juice. Lamaensky has established this fact in animals, from which he had extirpated the cœliac plexus. We must, therefore, admit an autonomy of the gastric vaso-motors, similar to that of the autochthonous ganglia of the heart and intestines. It is evidently by the intermediation of these inter-parietal plexuses that troubles of digestion of a reflex character are produced—for example, the sudden arrest of digestion through the influence of a violent emotion. We have in colic another functional trouble of the abdominal plexuses, one which is, in reality, a neuralgia of the cœliac plexus. Lead-colic is, in some respects, the type of this variety of neuralgia. Recently Harnack has published the results of his experimental researches on the physiological action of lead, from which it appears, in addition to other facts, that the metal excites the autonomous ganglia situated in the walls of the intestines. In man, this excitation is especially manifested by a generalized contraction of the intestinal canal, which explains the obstinate constipation and colics which are such constant symptoms of chronic saturnine intoxication. In animals, however, this intoxication occasions, on the contrary, profuse diarrhœa, for the reason that lead, by exciting the autonomous ganglia of the intestines, produces an exaggeration of the peristaltic movements of these organs.

---

[1] *Wiener Presse*, No. 20, 1876.

It is also to an excitation of the nervous plexuses which ramify in the walls of the excretory canals of the liver that are to be attributed the painful paroxysms known as hepatic colic. Here the agent of excitation is a biliary calculus, plugging up the choledic or cystic duct, and which, by reflex action, causes the contraction of the walls of this canal. A strong compression of the nervous plexuses which send numerous branches to the walls of the excretory ducts of the bile is thus produced. Accessions of nephritic colic are developed by an identical mechanism. And there is also a vesical neuralgia, characterized, at the same time, by pains and strangury, this last being the result of reflex spasm of the bladder, which is due to the pain seated in the walls of this organ.

The experiments instituted by Rochefontaine demonstrate that, if, in an animal, we tie at the same time the splenic artery and the nerves of the splenic plexus, the spleen becomes congested, while, on the contrary, it would seem as if the vessels should empty themselves of their contents. To explain this apparently paradoxical congestion, it is said that there is produced a true aspiration of blood to the splenic veins, by reason of the paralytic relaxation of the non-striated fibres of the spleen, caused by the ligation of the nerves distributed to that organ. However correct this interpretation may be, the experiment of Rochefontaine may, in a certain way, explain the development of splenic congestion in adynamic fevers, which are characterized in general by a marked atony of the nervous system, and in particular of the splanchnic nerves.

The greater number of the congestive phenomena, which have the uterus as their situation, take their point of departure in an irritation of the nervous ramifications which the genital plexus sends to the womb and its anexæ. It is the fact, also, as regards the more or less painful contractions of which this organ is the seat, either in the pregnant or non-pregnant condition.

Finally, the abdominal sympathetic, which, in the physiological state, gives an absolutely unconscious sensibility to the viscera, becomes the seat of extremely violent pains when it is deranged in its functions. These pains are especially apt to occur when the terminal ramifications of the visceral plexuses are compressed, either by the contraction or distention of the walls of a hollow organ, or by a wound or injury. Every one can recall in his own experience the intense pain caused by a blow on the epigastrium or on the testicles. These visceral plexuses are, besides, the point of departure for reflex phenomena, originating either at the places injured or at a distance. A painful sensation starting from the testicle leads, at first, to a contraction of the muscular tissue of the scrotum, with retraction of the testicles. When this pain reaches a sufficient degree of intensity, the walls of the abdomen, and of the several hollow viscera contained in the abdominal cavity, enter also into contraction. There may even, in certain cases, be developed general convulsions, with or without lipothymia, the result, doubtless, of the reflex contraction of the vessels of the nervous centres, and of the olighemia resulting therefrom. The painful irritations transmitted to the terminal ramifications of the splanchnic nerves cause also contractions of the abdominal vessels. Soon, however, these relax, and then there is a stagnation of blood in the abdominal vessels, through the formation of a true depot capable of holding the whole of the blood in motion, when their paralytic relaxation passes certain limits. Thus are explained the paleness of the face, and of the skin generally, the cyanosis, the coldness of the extremities, the phenomena of cerebro-spinal anæmia, and the smallness and rapidity of the pulse, that are observed in all cases of peritoneal irritation, and also in the coma and stupor which ensue on severe wounds and injuries—a state designated, by English and American surgeons, *shock*. Goetz has realized the conditions under the influence of which this state is developed, in his well-known experiment, which consists in striking the belly of a frog against the edge of a table. He has seen the enormous development of the abdominal vessels and the ischæmia of other regions which at once ensue, and which entirely explain the morbid phenomena observed in the similar instances to which we have called attention.

SYMMETRICAL GANGRENE OF THE EXTREMITIES.

Under the name of symmetrical gangrene of the extremities, M. Maurice Reynaud described for the first time, in 1862, a variety of gangrene which, since then, has been given a place in systems of nosology as a distinct morbid entity. It presents the curious feature of being developed independently of any lesion of the circulatory apparatus.

Generally, this affection is observed in young subjects, preferably in females, and with those who possess a neurotic diathesis. Cold, moral emotions, and troubles of menstruation, act sometimes as causes.

As a rule, the gangrene attacks symmetrically the lower extremities, more rarely the upper extremities ; sometimes, also, the nose and the ears.

In the beginning the patient feels, in the parts which are about to be the seat of the gangrene, a sensation of tumefaction, which coincides with the paleness of the skin at the same points. At other times, the skin of the extremities is covered with bluish-colored spots. These are the indications of the interference with the circulation which precedes the development of the gangrene. This local anæmia may, it is true, occur without there being any further advance. Then the parts primarily exsanguined become the seat of a temporary congestion, accompanied with more or less severe pains, before the circulation again becomes regular. The condition, in fact, resembles that induced by the local application of cold. This local anæmia may disappear and reappear many times ; but, when it is the prelude to the mortification of the parts affected, the skin becomes covered with phlyctenæ, dry, tense, like parchment, and assumes the black coloration peculiar to sphacelated tissues. The process of mortification is announced by extremely sharp pains, which the patients compare, ordinarily, to the sensations caused by burns.

Inspection of the limbs affected with this kind of gangrene shows the absence of all lesions capable of producing obstruction of the vessels distributed to them. There is neither thrombosis, embolism, atheroma, nor any alteration whatever of the walls of the vessels. Moreover, M. Maurice Raynaud has called attention to the fact of the persistence of the pulse in the extremities, the seat of the affection in question.

This circumstance indicates clearly that, if the circulation is insufficient for the proper nutrition of the tissues, it is not entirely abolished. To explain the development of the gangrene, it is sufficient to suppose the existence of a durable vascular spasm limited to the affected part ; and this is what M. Maurice Raynaud has done. For him, the symmetrical gangrene of the extremities and the local anæmia which is its immediate cause are the consequences of a spasm of the small vessels, due to an excitation of the vaso-motor nerves which innervate their walls. This spasm may be of a reflex order, having for its point of departure a peripheral excitation having its seat within the extremities threatened, or in some other organ—the uterus, for instance.

This excitation will be reflected by the vaso-motor centre situated in the bulb. Naturally, the production of this vascular spasm and its persistence under the influence of an occasional cause of slight importance suppose that the reflex centres of the cord are in a morbidly exaggerated state of excitability.

M. Vulpian, while admitting the theory of a local vascular spasm as the cause of symmetrical gangrene of the extremities, believes that it is useless to allege the implication of the vaso-motor centres in the production of this spasm. This eminent physiologist contends that the reflex vascular constriction which presides over the development of symmetrical gangrene of the extremities is produced only by the intermediation of the ganglia situated in the course of the vaso-motor fibres at a short distance from their terminations in the vascular walls. The symmetrical disposition of the gangrene is more in accordance with this theory than with that which makes it depend upon a derangement of the central innervation. In fact, "if the local anæmia of the extremities is so often symmetrical in the affection described by M. Raynaud, it is explained by the fact that it affects subjects in whom the local predisposition is due to a general modification of the economy, and ought to be nearly equal in homologous parts of the two sides of the body."[1]

M. Vulpian, moreover, contends that, if gangrene of vaso-constrictive origin is always symmetrical, it will be necessary to get rid of some facts which, from a pathological point of view, naturally are embraced under M. Raynaud's designation. Thus, in this connection, he recalls the instance, adduced by M. Gubler, of a gangrene limited to one of the toes, occurring in a young woman in whom there was no evidence of a closure of the arteries of the corresponding limb. It would evidently be illogical to abstract from the so-called symmetrical gangrene a case of similar pathogeny, merely because the gangrene was unilateral.

In a more recent work, M. Raynaud has published some cases of symmetrical gangrene of the extremities, in which the ophthalmoscope revealed the existence of a constriction in the central artery of the retina. This is, then, the basis for a new argument in support of the vaso-motor theory adopted by this author. As a practical consequence of the vaso-constriction theory, M. Raynaud recommends the use of descending galvanic currents applied to the vertebral column throughout its length. These currents have the effect of weakening the excito-motor power of the cord and bulb, and hence of combating the vascular spasm of central origin, which is the point of departure in symmetrical gangrene of the extremities.

In connection with the affection described for the first time by M. Raynaud under the name of symmetrical gangrene of the extremities, mention must be made of the condition referred to by M. Vulpian[2] as *symmetrical congestion of the extremities.*

The case observed was that of a patient in whom regularly every day there were accessions of pain and heat in all four extremities, especially the legs. "The skin became red and very hot, the arteries of the feet, the pulsations of which could scarcely be felt in the intervals, during the paroxysms beat with great force, and appeared to be dilated. There was at the same time a very painful feeling of tension, and walking, by aggravating these troubles, became impossible. The patient only found relief by plunging his feet and the lower part of his legs in cold water." M. Vulpian, with some reserve, is nevertheless disposed to see in this case an example of a symmetrical neurosis of the extremities, determining by reflex action the dilatation of the vessels of these parts.

---

[1] "Leçons sur l'appareil vaso-moteur," tome ii., p. 620.

[2] *Op. cit.*, tome ii., p. 623.

SECTION VI.

TOXIC DISEASES OF THE NERVOUS SYSTEM.

THERE are certain substances which, when taken into the body gradually and for a long time, manifest their poisonous influence more especially upon the nervous system. Among these, lead, alcohol, bromine, mercury, and arsenic, may be particularly mentioned. Several of these substances are used as slow poisons with criminal intent, others are habitually employed by many persons as stimulants, sedatives, or cosmetics, others are used in the arts, and hence enter the systems of those who are brought in contact with them, and some are prescribed in such doses in the treatment of disease as to produce upon the patient their characteristic physiological effects.

It seems important that the peculiar phenomena which these substances are capable of causing, with the *rationale* of their mode of action, and the treatment best adapted to obviate their deleterious effects, should receive some attention, and I shall therefore devote a few pages to their consideration.

CHAPTER I.

PLUMBISM

Symptoms.—The phenomena manifested in the nervous system, as consequences of lead-poisoning, are *lead-encephalopathy*, *paralysis*, *a spasmodic and painful affection called lead-colic*, *anæsthesia*, and *hyperæsthesia*.

a. Lead-Encephalopathy.—The symptoms referable to the brain, due to lead-poisoning, may be slight or severe. In the first case the patient suffers from headache, vertigo, and various other abnormal sensations, such as fullness, and constriction, and is at the same time incapable of much intellectual exertion without suffering an increase of his physical symptoms. His mind is irritable and depressed, and his sleep is usually disturbed with unpleasant dreams. The digestion is generally deranged, and the whole appearance may be cachectic. Tremor may exist, especially in the hands. It is generally not extensive, consisting ordinarily of slight tremulous movements, which, though present when the muscles are at rest, is more distinctly manifested when the muscles are put in voluntary action.

This condition may undergo no further development, but it is often the precursory state of the more severe form of the affection.

In the severe form the symptoms may be manifested by delirium, convulsions, or coma, or by any two or all of these phenomena. This last was the case in a patient, a master-plumber, in whose case I was consulted in the summer of 1873. The attack began with acute delirium, lasting several days, and then alternating with paroxysms of convulsions. The seizure ended, after about two weeks, with profound coma of forty-eight hours' duration.

In the delirious form the patient may either present the symptoms of acute mania with excitement, or there may be a melancholic condition present. In either case there are illusions, hallucinations, and delusions. After a variable period a remission generally takes place, and this may go on to a complete disappearance of the symptoms, or be succeeded by a renewed exacerbation.

In the convulsive form, the paroxysms may or not be marked by loss of consciousness. They may be limited to a particular part of the body, as the face, neck, or arms, or they may be general. They may present somewhat the characteristics of tetanus or of epilepsy, or of both these diseases. In some cases the seizures are not distinguishable from idiopathic epileptic attacks; the patient has tonic and clonic convulsions, froths at the mouth, bites the tongue, may evacuate his urine or fæces, and passes into a soporous condition. Or there may be repeated attacks succeeding each other with such rapidity as to constitute a *status epilepticus*.

In the comatose variety, the stupor is sometimes developed with great suddenness, but is not often so profound as to prevent occasional manifestations of partial sensibility. Thus, if the patient be spoken to in a loud voice he opens his eyes, or if the skin be pinched he withdraws the part or contorts the countenance.

The pupils are generally dilated and insensible to light, and the cheeks and lips are puffed out in expiration. If, in cases in which these symptoms occur, the gums be examined, a blue line running along their margins will be discovered. Sometimes the whole extent of the gums is tinged, but generally the discolored portion is the edge in contact with the teeth, and about a line or at most two lines in width. Besides the discoloration, the tissue of the gums becomes soft and spongy, and it may become absorbed, leaving the roots of the teeth exposed. All these changes are more marked in the lower than in the upper jaw.

The breath is usually of a peculiar odor, and, if what is called the lead-cachexia be present, the complexion is pale, the hair lustreless and dry, and the body emaciated. It not unfrequently happens that the individuals who suffer from lead-encephalopathy have also been the subjects of some one or more of the other manifestations of lead-poisoning.

b. Lead-Paralysis.—**Symptoms.**—Before the occurrence of paralysis, the patient has probably suffered from attacks of lead-colic, or some other affection due to lead-poisoning, though this is not invariably the case. The immediately precursory symptoms connected with the loss of power are slight numbness and tremors in the muscles of the upper extremities. Occasionally, the muscles of the trunk and lower extremities become involved in the trembling.

Ere long the patient observes that he has difficulty in extending the fingers or wrist, and that there is a general loss of strength in one or both hands. These symptoms go on increasing in severity, and eventually he loses the power to raise the hand or fingers. In extreme cases, the ability to extend the forearm, or to raise the arm from the side, is lost through the paralysis of the triceps and deltoid, or, as in a case before my clinique, in January, 1876, the biceps may be paralyzed. Occasionally, the extensors of the lower extremity are involved in the paralysis.

The predominance of the loss of power in the extensors has led to the idea that they alone are affected. The dropping of the hand, the flexion of the forearm on the arm, the hanging of the arm against the side of the body, and, when the lower extremity is affected, the inability to raise the toes so as to avoid striking them aginst the ground in walking, all give countenance to this supposition. But careful observation shows that the difference is merely one of degree, and that there is a very considerable loss of power in the flexor muscles. Indeed, of many cases of the disease that I have observed in hospital and private practice, I have never seen one in which the flexors were not implicated with the extensors.

Owing to the disuse of the muscles, atrophy takes place, and this is frequently exceedingly well marked, and, from the disturbance of the normal equilibrium between the several groups of muscles, contractions and distortions ensue. The circulation in the affected limbs becomes languid and weak, and painful swellings result in consequence.

It is generally supposed that the right arm is more apt to be affected than the left; such, however, does not appear to be the case

Thus, Tanquerel des Planches,[1] of seventy-nine cases in which the upper extremities were the seat of the paralysis, found both affected in fifty-one, the left twenty-three times, and the right twenty-four. Of thirty-two cases of lead-paralysis occurring in my own practice, the upper extremities were affected in all; in twenty-seven both limbs were the seat; and, of the remaining five, three were in the left, and two in the right. The left upper extremity was therefore affected thirty times, and the right twenty-nine.

In some cases, the muscles of respiration become very seriously paralyzed through the influence of lead, and death then soon takes place. In two of my cases there was aphonia, and in several the voice was materially weakened. Cases of hemiplegia, the result of lead-poisoning, have been observed by Stoll, Andral, and Tanquerel des Planches.

The electric sensibility and contractility are always greatly reduced in all cases of lead-paralysis. In the majority of cases, no faradaic current, which it is safe to employ, will produce contractions, and strong primary currents are necessary. The cutaneous sensibility is rarely impaired.

The saturnine cachexia is almost always present, and the blue line on the gums can readily be distinguished.

c. Lead-Colic.—This is probably the most common affection caused by the toxic influence of lead, and has been recognized from a very early period.

Symptoms.—Lead-colic is particularly characterized by the presence of pain, the apparent seat of which is at or near the umbilicus, although it may exist at the epigastrium, the hypogastrium, or some other part of the abdomen.

The character of the pain is somewhat peculiar, being a twisting sensation of great agony, which appears to revolve around the umbilicus. In some cases the distress of the patient is extreme, and he gives utterance to loud cries of anguish, and tosses himself about with the utmost violence. Nausea and vomiting are generally present, and the bowels are almost invariably constipated.

The respiration is ordinarily hurried and irregular, but the pulse, notwithstanding the physical and mental excitement, remains of its normal force, frequency, and rhythm, sometimes becoming markedly slower during the height of a paroxysm.

The abdomen is usually hard and retracted, especially during the height of a paroxysm.

Occasionally the abdomen is painful to the touch, and the suffering is aggravated by very slight pressure, but as a rule this is not the case. On the contrary, the pain, so far from being increased by pressure, is greatly relieved by it, especially if the force be exerted in a uniform and gradual manner. Patients often discover this fact for themselves, and will lie on the belly or press it with their hands, or beg that others will do so.

---

[1] "Traité des maladies de plomb," Paris, 1839, tome ii., p. 39.

The duration of a paroxysm is variable. It may last only a few minutes, or may be prolonged for an hour or more. A period of comparative calm then ensues, during which the exhausted patient may sleep a little, but his slumber is soon disturbed by another seizure, and this sequence may continue for several days. Paroxysms are more common and more severe during the night than the day, and sometimes the relation is observed with sidereal punctuality.

In consequence of the treatment adopted, or spontaneously, the series of attacks is broken, and the patient, for the time at least, recovers his ordinary state of health. It is exceedingly rare that death ensues from simple, uncomplicated lead-colic.

d. Lead-Anæsthesia.—Anæsthesia, as a condition due to the toxic influence of lead, may exist without complication with other manifestations, although such an event is not common. In the majority of cases it is the optic nerve which is affected, and as a consequence more or less complete blindness is produced. Some of the cases formerly reported were probably, as Stellwag[1] observes, simply instances of ciliary paralysis, but this author admits the existence of an organic affection of the nerve, terminating in atrophy, and recognizable by the ophthalmoscope.

Again, the anæsthesia may affect the skin of the trunk or extremities, or the muscles of these parts. It is developed generally with great rapidity, reaching its height in a few hours.

e. Lead-Hyperæsthesia.—The pains in the limbs and trunk are among the most common of the phenomena of lead-poisoning. The lower extremities are generally their seat, and by preference the flexures of the joints. Thus the groin and the popliteal space are favorite situations in the lower limbs; the axilla and bend of the elbow in the upper extremities. The back and thorax are also often affected, and sometimes the scalp, face, and neck.

The pains may be either of a dull aching character, acute, like the sensation from the thrust of a sharp instrument, or hot, as if a coal of fire were in contact with the part. They occur in paroxysms, and are apparently excited by cold, movements, or emotional disturbance. Occasionally there are spasmodic contractions of the muscles of the painful part, either *en masse*, singly, or in the form of fibrillary contractions.

Like the pains of lead-colic, they are generally relieved by steady and gradual pressure, but occasionally this is not the case, any kind of touch, light or heavy, causing an aggravation in their intensity.

---

[1] "A Treatise on the Diseases of the Eye," Hackley and Roosa's translation, New York, 1868, p. 668.

There is no increased heat of the painful region, no redness or swelling, and the pulse is generally normal.

In some cases the pains appear to be seated in the bones; usually, however, the skin and muscles seem to be their situation.

Causes.—The fact that such affections as those mentioned follow the introduction of lead into the system admits of no doubt. This introduction may take place through the stomach, the air-passages, or the skin. The two latter are the more common channels for contamination.

They are, of course, more frequently encountered among those who work in lead, such as lead-founders and smelters, the makers of white and red lead, painters, plumbers, printers, etc.; although they may occur among those who are only temporarily or accidentally exposed to the toxic influence. Thus, they may be caused by drinking water which has passed through lead pipes, or been kept in lead vessels, or by using tobacco which has been wrapped in lead-foil. Two cases in which paralysis was produced by the latter cause have happened in my experience,[1] and it is so common a cause that, in France, Belgium, and Prussia, strong laws have been passed against packing tobacco in lead. The use of hair-dyes containing lead is, I think, quite a common cause of plumbism. Three cases of paralysis and two of anæsthesia, in which this was the cause, have come under my observation, and I am inclined to think that a case in which there were vertigo, slight delirium, and one epileptic convulsion, owed its origination to the application of lead to the hair.

The employment of powders and enamels to the face is a not infrequent cause of plumbism in women, as most of those substances called cosmetics contain lead. Three cases of paralysis and one of pains in the body and limbs, caused by lead applied to the face, neck, and arms have occurred in my experience.

The use of plasters and lotions containing lead has also been known to give rise to plumbism.

The majority of cases, however, occur in this country in painters, probably for the reason that workers in white and red lead, though more exposed, are aware of their danger, and take effectual measures to prevent absorption.

Though the carbonate is probably the most actively poisonous preparation of lead, it is very certain that all forms—not even excepting the sulphate—are capable of producing the characteristic phenomena of plumbism.

Diagnosis.—The history of the case, including a knowledge of the occupation of the patient, or of his exposure to the action of lead, will generally prevent error of diagnosis in regard to any of the manifestations of plumbism. The presence of the peculiar cachexia and the existence of the blue line around the gums will tend still further to render the diagnosis accurate.

---

[1] *See* my translation of Meyer's "Electricity in its Relations to Practical Medicine," New York, 1870, p. 181, for reference to other cases.

Again, it has been ascertained, by the researches of Melsens, that the iodide of potassium has the faculty, when taken into the system, of decomposing the albuminates with which the lead is united, and of setting this substance free. It, then, at once appears in the urine, and can be detected by examination with sulphuretted hydrogen. A ready method is that proposed by Reeves. A piece of sulphide of potassium is inclosed in a piece of thin white linen, and suspended in a vessel containing the urine suspected to contain the lead set free by the previous administration of the iodide of potassium. It is left there for five or six minutes. If the urine contains any salt of lead, it is decomposed, and the metal is deposited on the linen in the form of the sulphuret, staining it of a dark, almost black, color.

As regards the several affections separately, it is to be remarked that most difficulty will be experienced relatively to the encephalopathy produced by lead. Jaccoud[1] has pointed out that in the condition of the bodily temperature we have an additional point toward discriminating between acute cerebro-spinal meningitis and the affection under notice. In the former the temperature rises to 104° Fahr., or even higher, while in the latter there is no augmentation, or at least a very slight rise.

In lead-paralysis the fact that the loss of power mainly affects the extensors, especially those of the hand, together with the antecedents of the patient, and the presence of other evidence of plumbism, will generally suffice to render the diagnosis certain. I have recently, however, had a case at my clinique at the University Medical College, in which there was some doubt. The patient had paralysis of the extensors of both wrists. Several weeks before its appearance he had broken his ankle, and had been obliged to walk on crutches. There was, therefore, a question as to whether the case was one of "crutch-paralysis," from pressure, or of lead-paralysis. The man was a laborer, and had never, to his knowledge, been exposed to lead in any way. But the facts that there was no anæsthesia, that the paralysis was greatly predominant in the extensors, and that the muscles of the arm above the elbow were not affected, decided me in concluding that the case was not one resulting from pressure on the brachial plexus. The further fact that there was a slight blue line visible along the gums convinced me that, notwithstanding the absence of any history of contamination by lead, the case was one of that disease. The patient was a beer-drinker, and might, I conceived, have become poisoned in that way, as had others in my experience.

In lead-colic the character of the pain and its situation may be of service in the formation of a diagnosis, but the main reliance must be upon the antecedents of the patient, and the coexisting evidences of plumbism to which attention has already been directed.

---

[1] "Leçons de clinique médicale," Paris, 1869, p. 492.

These circumstances are likewise what must govern us in lead-anæsthesia and hyperæsthesia.

Prognosis.—This is not unfavorable except as regards the cerebral manifestations, provided the patient can be submitted to proper treatment and removed from all exposure to lead-poisoning. Lead-encephalopathy is the most serious of all the forms of plumbism, and this is especially the case when there is a combination of delirium, convulsions, and coma. Of seventy-two cases observed by Tanquerel des Planches, sixteen were fatal. It was probably more apt to terminate in death in his day than now, when the hygienic and therapeutical relations of plumbism are better understood. Recovery ensued in all of the cases occurring in my experience.

In lead-paralysis the prospect of recovery depends altogether on the ability to produce contractions in the paralyzed muscles by electricity. If the induced current will effect them, the cure will be rapid; if the interrupted primary current is required, a longer time must elapse before success is attained; but, if the muscles will not react to either the induced or primary currents, a favorable result is not to be expected. The extent of the atrophy is also an important element in the prognosis.

In lead-colic, hyperæsthesia, and anæsthesia, the prospect of recovery is good, provided the necessary hygienic and therapeutical indications can be fulfilled.

Morbid Anatomy and Pathology.—Very little is known relative to the morbid anatomy of plumbic affections. In the several forms with which we are acquainted, the nervous system rarely presents evidences of any lesion which can be regarded as characteristic. In some cases of lead-encephalopathy, however, there has been found a flattened, indurated, and atrophied condition of the brain, and in others the indications of inflammation and softening. In the case of a painter who had suffered from repeated attacks of lead-colic, and who finally died with head-symptoms—delirium, epilepsy—reported by MM. Guéneau de Mussy and Lémaire, the post-mortem examination showed the existence of a large extravasation, which had broken through the cerebral tissue from the circumference to the fourth ventricle.

Gombault[1] recently reported a case of lead-paralysis, in which, on post-mortem examination, the spinal cord and the nerve-roots were found to be unaltered, but in some of the peripheral nerves the medullary substance was separated into granules, though the axis-cylinder was normal.

---

[1] *Archives de physiologie*, 1873.

Westphal[1] has discovered, in a case of lead-paralysis, a similar condition of the radial nerve. In this case the spinal cord and the nerve-roots were unchanged.

In a case of lead-poisoning in which there had been, during life, colic, vomiting, diarrhœa, and finally collapse, Kussmaul and Maier[2] found sclerosis of the cœliac and superior cervical ganglia of the sympathetic and periarteritis in the brain and spinal cord.

Lead has been detected, in cases of plumbism, in the tissue of the brain, spinal cord, and nerves. In fact, it appears to have a special affinity for the nerve-substance.

It is probable that, except in extreme cases, or in very exceptional instances, the changes in the brain, spinal cord, nerves, and sympathetic system are not such as are discoverable by our present means of research, just as are the alterations produced by opium, alcohol, hydrocyanic acid, strychnia, and other substances.

The muscles, in cases of lead-paralysis, have been examined by Andral,[3] Gendrin,[4] Tanquerel des Planches,[5] and others, and analogous results obtained. The fibres have been found to be pale and yellowish, to be friable, atrophied, and desiccated. I have repeatedly removed small portions with Duchenne's trocar, and have always found the transverse striæ disappearing, and fatty degeneration making its appearance.

The hypothesis, that the affection is, primarily, one of the muscles, is not supported by facts. Such a thing as muscular paralysis, independent of nervous derangement somewhere, is unknown in the whole range of pathology. And those cases of apparent loss of muscular irritability, resulting from certain poisons, adduced by Longet, Bernard, Mitchell, myself, and others, were simply instances in which the loss of nervous irritability took place from the periphery to the centre. The present state of our neurological knowledge is altogether against the idea of muscular irritability independent of the nerves. When a muscle is no longer capable of contracting, it is because the nerves are dead.

Facts, too, are against the notion that the lead acts by contact with the muscles, and the circumstance of the paralysis occurring so generally in the hands of painters, for instance, is adduced in proof. But we have seen that the left hand is just as frequently affected as the right, while it is certainly less in contact with the lead. Moreover, those cases of paralysis in the extensors of the hand which have resulted from hair-dyes and other cosmetics, are altogether against the hypothesis in question.

---

[1] *Archiv für Psychiatrie*, Band iv., 1874.

[2] *Deutsches Archiv für klin. Med.*, Band ix., H. 2.

[3] "Clinique Médicale," tome ii., p. 227.

[4] "Maladies de l'encéphale," par Abercrombie, traduction, second édition, p. 576

[5] *Op. cit.*, pp. 77, 144, 149.

In cases of lead-colic there appear to be no anatomical changes in the intestines which can be reasonably associated with the phenomena of the disease as their cause.

Treatment.—In the treatment of plumbism there are certain principles to be acted upon in all the affections embraced within its limits. One of these, the prophylaxis, belongs to the domain of hygiene, and therefore need not be here considered; the other, the removal of the lead from the system, demands our first care.

The researches of Melsens have shown that in the iodide of potassium we have an agent which separates the lead contained in the tissues from its combinations, and forms with it an iodide of lead, under which form it is excreted from the organism by the kidneys.

Some authors advise caution in the use of the iodide of potassium, on the ground that the resulting compound is very poisonous, and may produce highly-deleterious effects. In a great many cases of lead-paralysis and other consequences of lead-poisoning in which I have given the iodide, I have never seen the least untoward result, and I always use it in large doses from the beginning. In many cases the lead can be readily detected in the urine, and the blue line around the gums disappears quickly under its use. If there is great debility, or if the cachexia be marked, iron, quinine, and strychnia, may be employed with advantage.

In the treatment of lead-encephalopathy, the free administration of the iodide of potassium combined with the bromide affords the best prospect of success.

In attacks of lead-colic, the hypodermic injection of morphia, in doses sufficient to keep the pain in check while the iodide of potassium is doing its work, with an occasional purgative, will generally be all the treatment required.

But in lead-paralysis the loss of power remains, and would continue indefinitely, without the use of measures directed specially against it: chief among these is electricity. The induced current, if it will cause the muscles to contract, is to be preferred. Each paralyzed muscle must be acted on for two or three minutes every alternate day, so that for both upper extremities the duration of a *séance* would vary from a half to three-quarters of an hour. In ordinary cases two months will suffice to effect a cure.

But it often happens that the electric contractility of the paralyzed muscles is so completely abolished that the induced current is without effect. In such cases the primary interrupted current must be used, and continued till, as will eventually be the case, the induced current causes contractions. I have never seen a case in which the primary current would not produce contractions. One of the worst examples of the affection in question I ever saw was the patient who formed the subject of a clinical lecture to the class at the Bellevue Hospital Medical College.[1] His improvement under the circumstances was rapid, and he eventually was able to earn his living again. Induced currents of great power failed to produce contractions, and but for the use of the primary current he would have been incurable.

If the primary current fails to act on the muscles, success is out of the question.

In addition to electricity, frictions, kneading the muscles, and passive exercise, are useful. Contractions may be overcome by suitable prothetic apparatus.

In a case under the care of Prof. Sayre, and which I had the opportunity of seeing, the patient, a young lady, was able to play the piano—though paralyzed in both hands—by means of an admirable appliance devised by Dr. E. D. Hudson, of this city.

In the treatment of lead-anæsthesia and hyperæsthesia, the iodide of potassium conjoined with the use of the galvanic or faradaic current to the affected parts will generally prove sufficient to effect a cure.

CHAPTER II.

ALCOHOLISM.

ALCOHOLISM—under which term I do not now propose to embrace the condition called drunkenness, the immediate result of the ingestion of a large quantity of alcohol—is exhibited under two somewhat different forms. One of them is the permanent state which exists in persons who habitually imbibe excessive amounts of alcohol, and is known as chronic alcoholic intoxication or chronic alcoholism. The other is a paroxysm, the result of still greater excess, or the sudden stoppage of the stimulus to which the system has become habituated, and is designated by various names, such as delirium tremens, *mania a potu*, or more properly acute alcoholism.

a. Chronic Alcoholism.—The attention of the medical profession was first prominently directed to the subject of chronic alcoholism by Dr. Magnus Huss,[2] of Stockholm, in 1849. In my description of the disorder, I shall, to a great extent, avail myself of his thorough observations.

---

[1] *Journal of Psychological Medicine*, January, 1871, p. 43.

[2] Dr. Huss's work, being printed in Swedish, is to a great extent unread outside of Scandinavia. Two very excellent articles, embracing a full synopsis of his work, were published in the *British and Foreign Medico-Chirurgical Review*, in 1851 and 1852. I shall also draw largely from an address on "The Effects of Alcohol upon the Nervous System," which I delivered May 4, 1874, on assuming the presidency of the New York Neurological Society, and which was published in the *Psychological and Medico-Legal Journal*, for July, 1874.

Symptoms.—In one group of cases resulting from the long use of intoxicating liquors, the principal manifestations of the disease relate to the muscular system. Tremor and unsteadiness, especially of the upper extremities, are among the first symptoms. Subsequently the lower limbs are affected, and then the muscles of the trunk. These phenomena are most marked in the morning, before the patient has had his accustomed dram.

In other cases the tremor is not a very prominent feature, though, as far as my experience goes—and it is by no means inconsiderable—no patient with the disorder in question is free from a tremulous agitation of his muscles when he attempts to make a voluntary movement. But it may not be well marked, and, instead of it, the individual observes that he cannot hold things as well as he once did. Objects which he takes hold of fall from his hands without his being able to retain them. If he does exert himself to avoid this inconvenience, the hands are seized with an involuntary trembling, which he calls "nervousness," and which he endeavors to cure by fresh potations. From this feebléness or paresis the distance to paralysis is not great.

I had, not long ago, a case under my charge in which the patient, a gentleman of admitted eminence in his profession, clearly suffering from chronic alcoholism, could hold nothing in his hands unless he kept his eyes fixed upon them. The moment he ceased to look, the object fell to the ground. In the present treatise I have referred to several instances of this curious condition which were due to other causes.

The lower extremities eventually become affected, and the patient may entirely lose the power of locomotion. The nerves of sensation also become involved, and there are various abnormal feelings, constituting one or more of the forms of anæsthesia. Vertigo and dimness of vision may also be present.

This type of the disease Dr. Huss calls the paralytic.

In the next form, or the anæsthetic, the phenomena are more directly connected with perverted or lost sensibility from the outset. The extremities first become affected, and subsequently the central parts of the body. In the beginning the patient experiences a difficulty in determining from the feel the nature of the object he has laid hold of, or against which his foot may have struck. But in a more advanced stage he loses all sense of pain, and pins may be thrust into his skin, or a coal of fire dropped upon it without his experiencing any discomfort. With the anæsthesia there is always loss of motor power.

The æsthesiometer, the application of which instrument to practical medicine is of more recent date than Dr. Huss's observations, enables us to detect incipient loss of sensibility at a very early stage of the affection.

Symptoms connected with this category of cases which I have noticed, but which are not alluded to by Dr. Huss, are that the senses of sight, hearing, smell, and taste, are also often involved.

Another singular phenomenon which I have observed in these cases, which is referred to by Magnan,[1] and also quite recently by Virenque,[2] is that the loss of sensation involves only one lateral half of the body. This hemi-anæsthesia is met with in several other morbid conditions, notably as we have seen in hysteria. The other special senses are generally implicated. Thus the patient loses the sight of one eye; cannot hear with one ear; can taste the most strongly sapid substances with only one half of the tongue, and perceive the most penetrating odors with only one nostril. In one case cited by Magnan, the patient, who had long been addicted to the excessive use of alcoholic liquors, and subsequently to the use of absinthe, had hallucinations and delusions in addition to the hemi-anæsthesia, and, what is unusual, complete loss of sensation in the cornea of one eye, although tears were excited in both eyes when the affected one was touched by the finger.

In the third form of chronic alcoholism, convulsions constitute a prominent feature, though they are not generally among the first symptoms. I have, however, witnessed several cases in which epileptiform seizures were the immediate and direct consequence of the excessive use of alcoholic liquors, and in which there had been no well-marked premonitory symptoms. But in the great majority of instances there are derangements of motility and of sensibility, such as have just been described, and then the gradual supervention of convulsive jerkings of the muscles, similar to those which occur in convulsive tremor and chorea, combined with painful tonic contractions or cramps. After a time the spasms are accompanied with loss of consciousness, and hence are more truly epileptic in character. Dr. Huss noticed that as the condition of chronic alcoholism became more profound there was a tendency toward the disappearance of the convulsions, and that at last they ceased entirely.

In the next and last variety of the affection there is a general hyperæsthetic condition of the skin and other special organs of the senses. The least touch causes intense pain; bright lights are unendurable, and even the diffused light of a moderately illuminated room is painful. Very gentle noises cause great discomfort, and loud sounds are agonizing. Even the smell and taste are exaggerated, and occasionally perverted to the extent of illusions.

In whatever form chronic alcoholic intoxication may manifest itself, there are occasionally notable symptoms present which do not constitute ordinary features of the disease. Thus there may be double vision, from paralysis of one of the ocular muscles, usually the internal rectus, in which case there is ptosis also; or the muscles concerned in articulation are involved, and speech becomes imperfect or impossible; or those by which swallowing is effected are paralyzed, or there is violent palpitation of the heart, or intense neuralgic pain in one or more parts of the body. To touch on all these complications would require more space than I have at my disposal. But the mental symptoms which form more or less prominent characteristics of all cases of chronic alcoholism require a somewhat extended notice. The perceptions, the emotions, the intellect, and the will, are all implicated to a greater or less extent. Attention has already been called to the aberrations of the perceptions constituting illusions and hallucinations. The emotions assume an undue prominence, especially those of a sorrowful character, and thus the individual becomes maudlin, a condition which I should describe as consisting in a disposition to lament and shed tears over imaginary or greatly exaggerated griefs. It is rarely the case in my experience that the subject of chronic alcoholism is changed from a peaceable to a quarrelsome person, or from a timid to a brave one. The alteration is almost always in the other direction. At the same time it is not to be denied that individuals, whose passions are vicious and not held in complete subjection, are rendered still more vicious and uncontrollable by chronic alcoholism. Perhaps the most characteristic feature, as regards the emotions which persons suffering from the disease in question exhibit, is irritability of temper. This is shown in the fact that slight circumstances, which in a state of health would cause no annoyance, now give rise to great vexation. At the same time, though there is not, as I have said, much tendency to quarrelsomeness, there is nevertheless a proneness to take offense, and to regard, as slights and insults, acts which have no bearing in that direction.

---

[1] "De l'alcoolisme, des diverses formes, du délire alcoolique et de leur traitement," Paris, 1874.

[2] "De la perte de la sensibilité générale et spéciale d'un côté du corps," etc., Paris, 1874.

Again, there is intense melancholy, without the existence of delusions, and during which the individual may attempt suicide; or there may be indefinable fear, despair, terror, or shame, leading to the perpetration of self-destruction.

The more purely intellectual qualities of the mind rarely escape being involved in the general disturbance. The power of application, of appreciating the bearing of facts, of drawing distinctions, of exercising the judgment aright, and even of comprehension, are all more or less impaired. The sense of right and justice which the individual may have had is so weakened or destroyed that he will lie, steal, murder, or commit other outrages, even when there is no provocation. Indeed, the existence of motive is generally a counteracting circumstance.

The memory is among the first faculties to suffer.

But in addition to these evidences of mental deterioration there may be actual aberration of mind, as shown by the existence of delusions.

These are generally of a depressing character, and may or may not have their origin in false perceptions of the senses. These delusions may prompt to suicide or other act of violence.

The will is always lessened in force and activity. The ability to determine between two or more alternatives, to resolve to act when action is necessary, no longer exists in full power, and the individual becomes vacillating, uncertain, the prey to his various passions, and to the influence of vicious counsels.

With these troubles of the mind there are almost invariably headache, vertigo, and persistent wakefulness, all of which give evidence of the extent to which the nervous system is affected.

All writers of systematic treatises upon insanity have called attention to the frequency with which mental aberration is caused by the excessive use of alcoholic liquors, but, in a recent monograph, M. Marfaing[1] has given some interesting data relative to the characteristics of the insanity produced by alcoholism. Thus, he has observed that the hallucinations and delusions are almost always of a painful character. The patient sees frightful or repulsive objects, armed men or horrible animals; he sees persons lying in wait for him, or a thousand obstacles are interposed between him and his desires; he hears menacing voices, and the supplications of his friends for help from dangers which encompass them.

Occasionally, however, the imaginings are of a more pleasant character. He is surrounded with flowers and fountains ; beautiful women are his companions, and, though his generative power may be entirely extinct, he brags of his conquests, and the favors which are showered upon him.

Another characteristic of the hallucinations and delusions of the mania of alcoholism is, their changeability. Scarcely has he expressed one delirious conception when another is uttered, and so on for days at a time.

A somewhat peculiar variety of chronic alcoholism is that produced by the drinking of absinthe, a habit which prevails to a great extent in France, and which, though barely naturalized in this country, has a large and increasing number of votaries.

The condition in question has been well studied by M. Magnan[2] by experiments on the lower animals as well as by observations in man. The main fact appears to be that absinthe has an especial proclivity to produce epileptic convulsions, in addition to causing the other phenomena due to the highly-concentrated alcohol it contains.

Death may ensue in chronic alcoholism, from the accompanying morbid conditions, induced in the brain or other parts of the nervous system; from exhaustion, owing to the direct effects of the poison, or to the inability of the stomach to digest, and the assimilative organs to appropriate the food taken; or, as is commonly the case, from the supervention of some intercurrent affection to which, owing to the depressed condition of the system, the patient is particularly liable.

---

[1] "De l'alcoolisme considéré dans ses rapports avec l'aliénation mentale," Paris, 1875.

[2] "Étude expérimentale et clinique sur l'alcoolisme," Paris, 1871 ; also "De l'alcoolisme," Paris, 1874.

b. Acute Alcoholic Intoxication, Delirium Tremens. **Symptoms.**—Among the first symptoms of acute alcoholism, gastric and intestinal derangements are to be noticed. Thus there are anorexia, nausea, and vomiting, especially in the morning, and either diarrhœa or obstinate constipation, and the tongue is furred and dry. The pulse is usually rapid and feeble, the skin cold and clammy, and the general powers of the system much reduced. The sleep is deficient in amount and is disturbed by frightful dreams, and there are often vertigo, headache, and confusion of ideas.

At a very early period tremor is present, and is especially manifested in the tongue, which, when protruded from the mouth, cannot be held steady, and the continual action of which is further shown in the defective articulation which always exists. The upper extremities and sometimes the head are also the seat of tremulous movements.

These symptoms gradually increase in intensity, and other phenomena are soon developed. The countenance assumes a wild expression, the manner becomes hurried and anxious, the illusions, hallucinations, and delusions, become more vivid, and they are almost invariably of a terrifying character. Frightful objects, such as reptiles, demons, and other horrible figures, are perceived, and the patient covers his head in the bedclothes in the vain endeavor to shut out the sight of them, or may even commit suicide in the effort to escape from the imaginary dangers which threaten him. Hallucinations of the other senses are also sometimes present. The temper becomes still more irritable, and the motility is increased to an extreme degree. Sleep is no longer possible, and day and night the visions and delusions are ever present in some form or other. The body becomes hot, but the extremities still remain cold and clammy. The pulse ranges from 100 to 120 or more, and is small and weak. The urine is scanty and high-colored, the bowels constipated.

During all this period the patient talks incessantly, generally with reference to his hallucinations and delusions. These latter, though well marked, and constant, are, like his erroneous perceptions, changeable; and it rarely happens that they cannot, for the moment at least, be dissipated by a few words from those around.

The pupils are usually strongly contracted, and if the fundus of the eye be examined with the ophthalmoscope, the disk and retina will be found congested. Dr. Clifford Allbutt[1] states that he makes it a rule to examine the fundus in all cases of delirium tremens which come under his observation, and that in the great majority of cases he finds congestion and opalescence of the disk, and full retinal veins.

---

[1] "On the Use of the Ophthalmoscope in Diseases of the Nervous System," London and New York, 1871, p. 258.

In some cases—especially in those which are the direct result of the excessive use of alcoholic liquors, and not the consequence of a sudden deprivation of an accustomed stimulus—convulsions of an epileptiform character may occur; usually these are repeated again and again. Death may take place during their continuance, and they always add greatly to the gravity of the situation.

An attack of acute alcoholism lasts ordinarily for from three to five days. If recovery is to ensue the patient obtains a little sleep, and awakes with a decided mitigation in the violence of all his symptoms. If, on the contrary, death is to result, his physical powers become rapidly exhausted, the delirium becomes low and muttering, he picks at the bedclothes, he passes into a state of coma, the pulse rises still higher in frequency, while it becomes correspondingly weaker, the bodily temperature falls, and he gradually sinks or dies from a renewal of the convulsive seizures.

Causes.—Though the abuse of alcohol as a beverage is the essential cause of alcoholism, chronic or acute, it is not to be supposed that these conditions are induced in all persons who use alcoholic liquors to excess. Some individuals are not only able to indulge to an extreme degree with impunity, but may even live to old age in the enjoyment of apparent good health. Indeed, when Huss published the results of his observations, it was strongly questioned whether the symptoms which he had noticed were not due to the impurities which the whiskey generally used by the lower classes in Sweden is known to contain, rather than to alcohol. Huss admits that since liquor made from potatoes came into use, and especially since it has been distilled from rotten potatoes, chronic alcoholism has become much more frequent. This was attributed to the fusel-oil and a peculiar substance called *stick*; but it was ascertained that, though these substances may have aggravated the symptoms, they were, in the main, produced by the alcohol. Many will doubtless call to mind that in this country a like charge has been made against fusel-oil, and that even strychnia has had the reputation of poisoning whiskey and inducing most of the evil effects of excessive alcoholic potations.

It is very certain, however, that alcoholic intoxication very rarely, if ever, ensues on the moderate use of the light German or French wines, or of those made in this country, when they are not fortified by the subsequent addition of spirit, and that it is still less apt to occur from the temperate use of malt liquors.

In those countries in which wine or beer is the chief alcoholic beverage, the peculiar conditions which have been described are rarely met with. Thus Niemeyer omitted from the earlier editions of his work on the "Practice of Medicine" all reference to either chronic or acute alcoholism, and a chapter was afterward specially added in order to render the work more useful to American and English physicians, for whom it was translated by Dr. C. E. Hackley, of this city. In France, also, before the recent increase in the consumption of the stronger alcoholic liquors and absinthe, neither form of the affection under notice had attracted much attention. Marfaing begins his monograph, to which reference has already been made, with the statement that previous to the last twenty-five years alcoholism was hardly known. But in the northern European countries, in Great Britain, and in the United States, where whiskey, gin, rum, and brandy, have been the more common forms under which alcohol has been ingested, delirium tremens has always been a prominent disease, and the chronic form doubtless existed long before Huss pointed out the features by which it was to be recognized.

It appears, therefore, that what are called the spirituous liquors are more powerful in causing alcoholism than either the malt or vinous. This is probably due to the facts that more alcohol is imbibed with the former than the latter, more than can promptly be eliminated, and that, owing to its concentrated form, greater derangement of the tissues, with which it comes in contact, is produced. It is thus with alcohol as with all other powerful agents taken into the system.

That acute alcoholism or delirium tremens results directly from the excessive ingestion of alcohol is admitted by all writers on the subject, but they are not so generally agreed that it may ensue indirectly from such excessive use, by the individual being suddenly deprived of the accustomed stimulus. Thus Aitken[1] denies in very positive terms that delirium tremens may occur as a consequence of cessation from drinking, but to my mind any one who has seen the disease in soldiers, sailors, or prisoners, will be slow to confirm his statements. I have frequently seen delirium tremens occur in soldiers whose debauches have been suddenly interrupted by confinement in the guard-house, and I am quite sure that most army, and navy, and prison medical officers have had similar experience. Watson,[2] on the other hand, assigns no other cause than that the "habitual stimulus has been diminished or abandoned;" but he subsequently, without seeming to notice the bearing of the case, refers to an instance in which the patient was constantly under the influence of alcoholic liquor. Dr. Flint,[3] however, distinctly recognizes this dual causation, but the fact does not appear to influence his views of pathology or treatment.

---

[1] "The Science and Practice of Medicine," third American edition, vol. ii., p. 847, Philadelphia, 1872.

[2] "Lectures on the Principles and Practice of Physic," American edition, Philadelphia, 1872, vol. i., p. 847.

[3] "A Treatise on the Principles and Practice of Medicine," third edition, Philadelphia, 1868, p. 735.

The one form occurs at the height of an alcoholic debauch; the other results when the system, habituated to large and repeated doses of alcohol, is suddenly deprived of a stimulus to which it has become thoroughly habituated. We see a like condition induced in those who, having become accustomed to the ingestion of opium, suddenly or too rapidly leave off the use of the drug.

In their therapeutical relations the distinction between these two modes of causation is, as we shall hereafter see, important.

Diagnosis.—The clinical history as well as the peculiar symptoms will prevent any mistake being made relative to the real character of a case of alcoholic intoxication, either chronic or acute.

Prognosis.—The chronic form is generally successfully treated if the patient can be made to abstain from the further use of alcohol. A paroxysm of the acute form is also usually recovered from, provided there have not been many previous attacks. The occurrence of convulsions is, however, a serious complication, and almost invariably cases in which they take place terminate fatally. If the patient abstains from the further excessive use of alcohol, it is not at all probable that other attacks will ensue.

Of course, these remarks refer to alcoholism, and not to the lesions in the stomach, liver, intestines, heart, and other organs, which may have resulted from the abuse of alcoholic liquors, but which are not directly connected with the nervous system.

Morbid Anatomy and Pathology.—The most common patho-anatomical condition of the nervous system met with in cases of alcoholism, chronic or acute, is congestion of the cerebral meninges and of the substance of the brain. This alteration is especially liable to affect the vertical surface. An effusion of serum is a general concomitant, particularly in the acute form of the disease—and this may be either in the subarachnoid space or in the ventricles. At a later period, if the excesses be continued, the dura mater may become chronically congested, and eventually pachymeningitis and hæmatoma are developed.

Or the repeated or continual congestion of the pia mater and arachnoid may result in the production of a chronic inflammatory process, attended with thickening and opalescence of these membranes. The vessels, especially the veins, are gorged with blood, and there may be various morbid products, such as serum, pus, or sero-pus effused.

The brain, however, presents the most characteristic alterations. These appear to be the result of irritation and degeneration, the latter process consisting of a granular or fatty disintegration of the cerebral tissue, generally most marked in the cortical substance.

Dr. John C. Peters,[1] of New York, was among the first to make careful and systematic observations of the post-mortem appearance of individuals who had died from the excessive use of ardent spirits. As regards the brain, he found that "invariably there was present more or less congestion of the scalp and of the membranes of the brain, with considerable serous effusion under the arachnoid, while the substance of the brain was unusually white and firm, as if it had lain in alcohol for an hour or two, and the ventricles were quite empty. In not more than eight or ten instances did we find more red spots upon the cut surface of the brain than usual. The peculiar firmness of the brain was noticed several times, even when decomposition of the rest of the body had made considerable advance."

---

[1] "On the Pathological Effects of Alcohol," *New York Journal of Medicine*, vol. iii, 1844, p. 335.

Such changes as are described cannot result entirely from congestion, but must be ascribed, in great part, to the direct action by contact of alcohol on the brain-substance. It will presently be shown how strong is the affinity of alcohol for this tissue. As Carpenter[1] remarks, alcohol passes into the brain and changes both its chemical and physical properties. It would be strange indeed, therefore, if with alteration of structure there were not also aberrations of function.

The experiments of Dr. Percy[2] have often been brought forward as proving something in regard to alcohol which was not true of any other substance. This observer injected strong alcohol into the stomachs of dogs. The quantity varied from two to six ounces. Death followed, and upon examining the blood and brain for alcohol it was always found. The presence of alcohol in the blood and brain, to those who look superficially or ignorantly at the matter, has rather a horrible aspect; but when we know that there is no substance capable of being absorbed by the stomach and intestines which cannot also, by proper means, be detected in the blood and viscera, the subject loses much of its striking character. Dr. Percy used alcohol of 850° specific gravity, which represents a mixture containing about eighty per cent. of absolute alcohol. As the strongest brandy and whiskey contain but about fifty-four per cent. of alcohol, the concentrated character of the liquor used by Dr. Percy is at once seen. In one case six ounces were injected into the stomach of a dog, a quantity amply sufficient to cause death in an adult man.

Many other physiologists have detected alcohol in the blood and viscera of animals after its ingestion into the stomach.

I have several times performed experiments with reference to this point, and have never failed to recognize the presence of alcohol in the blood, brain, the stomach, expired air, and urine of dogs to which I had administered strong alcohol; but, when using liquors containing from eight to fifteen per cent. of alcohol, such as the German, French, and

---

[1] "On the Use and Abuse of Alcoholic Liquors in Health and Disease," London, 1870.

[2] "An Experimental Inquiry concerning the Presence of Alcohol in the Ventricles of the Brain," etc., London, 1839.

Spanish wines, I have never been able to find it in the solids, though detecting it readily in the products of respiration.

It is not to be doubted, therefore, that alcohol, like other substances, is absorbed into the blood, and exerts its influence on the system through the medium of this fluid.

Pure alcohol is a violent poison. In the dose of less than one ounce I have seen it cause death in a medium-sized dog, and many cases are on record of fatal effects being immediately produced in the human subject after comparatively small quantities have been swallowed. When diluted, its effects are not so rapidly manifested, and from this form, when taken in sufficient quantity, the condition known as intoxication is produced. Previous to this point being reached the nervous and circulatory systems become excited, the mental faculties are more active, the heart beats fuller and more rapidly, the face becomes flushed, and the senses are rendered more acute in their operation. If now the further ingestion be stopped, the organism soon returns to its former condition, without any feeling of depression being experienced; but, if the potations are continued, the complete command of the faculties is lost, and a condition of temporary insanity is produced. If further quantities be imbibed, a state of prostration, marked by coma and complete abolition of the power of sensation and motion, follows. Such is a brief outline of the obvious symptoms which ensue upon the use of alcoholic liquors in considerable quantities. When taken in amounts less than are sufficient to induce any marked effect upon the circulatory and nervous systems, there is, nevertheless, an influence which is felt by the individual, and which is mildly excitatory of the mental and intellectual faculties.

The very important physiological relations of alcohol scarcely come within the scope of this treatise; but the pathological conditions which result from it are of importance in the present connection, and may therefore profitably engage a share of our attention.

The general action of a large dose of this substance is shown in the following experiment:

I caused a dog to take into its stomach three ounces of strong alcohol, diluted with a corresponding quantity of water. Immediately on receiving it, the animal retired to a corner of the room and lay down. At the end of five minutes I endeavored to make it walk about the apartment, but it did so with evident reluctance, though up to this time the gait was not staggering. I should have stated that I detected alcohol in the expired air in forty-eight seconds after administering the liquid.

After eight minutes the dog walked with some difficulty, and on carefully examining the gait I found that the posterior extremities were beginning to be paralyzed. This paralysis gradually increased, the gait became more and more staggering, and at the end of fourteen minutes the animal could no longer stand. The paralysis had now reached the anterior extremities.

Sensibility was still present, though evidently lessened in acuteness; loud noises were perceived, and the eyes were involuntarily closed when the motion of striking was made before them. The respiration was hurried, and the action of the heart was greatly accelerated.

The pupils were at first contracted, but became dilated in about fifteen minutes, and remained in that condition throughout the experiment.

In thirty minutes the animal was in a state of profound coma. Sensibility, even of the cornea, was abolished; the limbs were in a state of complete resolution; the respiration was hurried; the heart beat rapidly but feebly; the urine and fæces passed involuntarily, and the temperature, as indicated by a thermometer placed in the rectum, had fallen from 101° Fahr., which it was before the ingestion of the alcohol, to 98.5° Fahr.

The animal remained in a comatose condition, and died one hour and twenty-two minutes after the ingestion of the alcohol.

In this experiment the alcohol was administered in such a large dose that the period of excitation, which generally follows in a few minutes, was masked or altogether prevented. In the following experiment, the quantity was smaller, and the sequence of phenomena was more regular.

I introduced into the stomach of a large dog one ounce of alcohol, diluted as before.

Nothing occurred worthy of notice during the first five minutes. Then the heart was accelerated, as was also the respiration, and the pupils became contracted. Sensibility and the power of motion were unaffected.

In twelve minutes the gait of the animal became uncertain, the limbs were lifted higher than was natural, and the body swayed from side to side, and occasionally strong efforts had to be made to maintain the erect position. The pupils were still contracted, and sensibility appeared to be intact.

This condition lasted twenty-two minutes, and then the pupils began to dilate. The posterior extremities were so far weakened as to render locomotion impossible, and the sensibility of the posterior parts of the body was materially impaired; the respiration was very irregular, sometimes being quite rapid, then ceasing for several seconds, and then becoming slow. The pulse was still rapid, but weaker than at first. In a little less than an hour the animal was in a state of light coma, which lasted about twenty minutes. Recovery took place gradually, the phenomena of intoxication disappearing in an inverse order to their supervention.

Observation of the symptoms which ensue when alcohol in sufficient quantity is given to animals shows that the condition of intoxication may, as Marvaud[1] proposes, be divided into three periods or stages:

---

[1] "L'alcohol: son action physiologique," etc., Paris, 1872, p. 28.

1. *Period of Excitation.*—Uncertainty in the movements, acceleration of pulse and of respiration, contraction of the pupils.

2. *Period of Perversion.*—Muscular paralysis, beginning in the posterior extremities, irregularity of pulse and of respiration, dilatation of the pupils.

3. *Period of Collapse.*—Complete paralysis of motion, anæsthesia, feebleness of the pulse and of respiration, stoppage of respiration and of the heart's action, death.

Now, I was desirous of knowing how much of this condition was due to the presence of alcohol in the brain, and how much to disturbance in the quantity of blood normally present in this organ. In other words, I wished to ascertain whether alcohol increased or diminished the amount of blood circulating within the cranium. For this purpose I performed the following experiment:

I trephined a dog, and secured a cephalohæmometer into the opening made by the trephine in the skull. I then administered an ounce of alcohol, diluted as in the previous experiment. In fifty seconds I detected alcohol in the expired air; in four and a half minutes the respiration was accelerated, the action of the heart became more rapid and strong, and the pupils were beginning to contract. Still there was no increase in the intracranial pressure, and I therefore knew that up to this time the amount of blood in the brain had not been increased. In six minutes and a half the dog's gait was staggering, and, though his movements were uncertain, there was no paralysis. The intracranial pressure was still unaltered.

The fluid remained stationary in the tube of the instrument till seventeen minutes had elapsed. Then it began to rise slowly, and, with this increase in the intracranial pressure, paralysis of the posterior extremities supervened. As the amount of blood contained in the cranium became greater, the paralysis extended, the pupils dilated, and coma ensued. The return to sensibility and the power of motion was attended with a diminution of the intracranial pressure, and was probably directly dependent thereon.

I repeated this very instructive experiment twice with similar results.

The deductions to be made from them are, that the first symptoms which result from the ingestion of alcohol are due to the presence of this substance in the brain, while the latter phenomena are, in part at least, the results of cerebral congestion.

NOTE.—In these and other experiments detailed in this chapter, the presence of alcohol in the expired air was determined by causing the breath to pass through a solution of bichromate of potash in sulphuric acid, a test suggested by Masing,[1] and not by Lallemand, Perrin, and Duroy, as generally supposed.

---

[1] "De mutationibus spiritus vini in corpus ingesti," 1854.

In man a like sequence is observed. A single glass of wine induces an exhilaration and activity of mind before there is any evidence of an increase in the amount of blood circulating in the cerebral blood-vessels. In several subjects particularly sensitive to the action of alcohol, I have observed that the flushing of the face and increased vascularity of the fundus of the eye, as shown by the ophthalmoscope, were second in order of occurrence to others indicating mental excitement.

But, as is well known, the immediate effects of a large quantity of alcohol, when taken into the human stomach, are not limited to mental excitement and flushing of the face. It does not come within the scope of this chapter to consider all of them ; but so far as the nervous system is concerned they properly come under notice.

Lévy[1] divides the phenomena of alcoholic intoxication, as they relate to the nervous system, into three stages : excitement, perturbation, and destruction of the functions of the brain and spinal cord. The stage of excitement is characterized by a sensation of heat in the skin of the whole body and by redness of the face. The eyes appear to be larger and more brilliant, the ideas flow more readily, the tendency to talk is generally increased, but the articulation is usually not so distinct and exact as is natural. The disposition becomes more generous, and perhaps more reckless as to consequences, although the bounds of propriety of conduct and truth of expression are not exceeded.

Occasionally a different set of symptoms results. The individual, from being naturally talkative, becomes taciturn and stolid, and a generous disposition is changed to one of which churlishness and selfishness are the chief features.

If the quantity of alcohol taken has been small, or if the individual now ceases to drink it, the subsequent stages do not supervene, and the equilibrium is soon restored without the occurrence of any abnormal condition. But, if the amount ingested has been large, or if the potations are continued, the second stage, that of perturbation, ensues.

There are now vertigo, disturbances of sight—such as result from paralysis of one or more of the ocular muscles, and giving rise to double vision—contraction of the pupils, noises in the ears, and increased redness of the face. The sense of taste becomes weakened, the voice loses its natural inflections and becomes rough and monotonous, and the articulation is indistinct from partial paralysis and defective coördination of the muscles of speech.

The gait, from like causes, becomes weak and uncertain, and hence, if the individual attempts to walk, he staggers. The movements of the upper extremities are irregular, and often exhibit marked tremor like that which constitutes so prominent a feature of paralysis agitans, or of some of the forms of sclerosis affecting the brain and spinal cord.

[1] "Traité d'hygiène," tome ii., Paris, 1862, p. 63.

Still greater alterations from the normal standard are shown in the mind than in other manifestations of nervous action. The most striking change occurs with the emotions, which generally assume an undue prominence and dominate over other of the mental faculties. And it not infrequently happens that the feeling which is most conspicuous is the very opposite of that which is natural to the individual. Thus the brave man becomes cowardly, the timid courageous, the peaceable quarrelsome, the modest shameless, etc. Usually, however, the emotions, which the subject in his normal condition is able to control and to keep in proper subordination to the intellect and will, become exaggerated, and are no longer held in subjection. It therefore happens that, when this stage of alcoholic intoxication is reached, the individual, who while in his natural state is high-toned and spirited, is ready to take offense and engage in quarrels upon the slightest provocation, and often when no cause for his emotion and conduct exists. It is in this stage that outrages against the law are most apt to occur.

The more purely intellectual part of the mind does not escape. The judgment is weakened, the memory impaired, the imagination exalted or perverted, and delusions, often having their origin in disordered sensations, and often arising in the mind without any accompanying illusion or hallucination, may assume the government of the thoughts and actions. The ability to grasp the details of a subject, and to comprehend them, is greatly injured, or even altogether destroyed, and hence study or continuous and systematic thought is no longer possible.

In the third stage the full action of the alcohol is attained. The mental, sensorial, and motor functions are more or less completely abolished, and death, generally the direct result of suspension of the respiratory movements, may ensue. When this degree of alcoholic intoxication is at its height, the individual is dead to all external impressions. Boiling water may be poured on his body, but he does not feel it; speech is impossible; the sphincters are relaxed, allowing the contents of the bowels and bladder to escape; the pupils are largely dilated; the breathing is slow, heavy, and often stertorous; the face is swollen and purple from the circulation of non-oxygenized blood through the vessels; and the power of thought is extinct. With the exception of that part of the cerebro-spinal axis which presides over the functions of respiration and circulation, the individual is to all appearance dead. It not infrequently happens that this region is so fully affected that life is abolished.

Such are the immediate effects of large quantities of alcohol when ingested into the human stomach. No one can fail to observe that most of the remarkable phenomena which follow on the administration of this liquid are connected directly or indirectly with the nervous system. Indeed, experiments performed upon animals, with reference to this point, as well as careful observation of the effects of alcohol on the human organism, show that this substance has a signal affinity for the nervous tissue, and that it is even capable of acting powerfully on the brain, the spinal cord, and the sympathetic system, without the intermediation of the blood. Instances are on record, and I have myself witnessed one such, in which a large quantity of alcoholic liquor taken into the stomach has produced death in a few minutes; and Orfila[1] cites a case in which a man died immediately from the effects of an excessive dose of brandy. I have several times killed rabbits in less than a minute by introducing an ounce of pure alcohol into the stomach. In such cases the action is not exerted through the medium of the blood, but directly on the sympathetic system or medulla oblongata by the terminal nerve-branches in the stomach. Indeed, if, as I have frequently done, a like amount of alcohol be injected into the blood directly, death does not ensue with so great a degree of rapidity.

Marcet[2] says:

"By experimenting on frogs I have shown, in a paper read to the British Association, in 1859, that a sudden temporary suspension of sensibility or shock is occasionally brought on when the hind-legs of these animals are suddenly immersed in strong alcohol; and I have obtained positive proof that this phenomenon is due to an influence exerted exclusively on the extremities of the nerves supplying those limbs, by observing this same effect to take place after the circulation of the parts in contact with alcohol had been entirely arrested. When, on the contrary, the nerves of the limb immersed in alcohol were severed from their centre, the circulation being left undisturbed, a shock never happened. In the experiments in question it was obvious that the sudden occurrence of insensibility or anæsthesia was due to an action of the alcoholic fluid on the extremities of the cerebro-spinal nerves, which action had been transmitted by these nerves to the brain; the phenomena of reflex action continued, for the respiration appeared unimpaired, and after the lapse of some minutes the shock passed off with a return of sensibility, although the frog's hind-legs had not been removed from the alcohol."

I have repeated Marcet's experiments, with every possible precaution to guard against fallacy, and am satisfied that his conclusions are correct. In one experiment I divided all the tissues of both posterior limbs of a large frog, except the sciatic nerves. I then placed small slips of thin glass under these nerves, and moistened them with a few drops of pure olive-oil, so as to prevent the alcohol acting by imbibition. I then plunged both limbs up to the thighs in absolute alcohol. Shock ensued in eleven seconds, and lasted about five minutes. During its continuance the animal was insensible and anæsthetic.

---

[1] "Toxicologie," tome ii., p. 528.

[2] "Chronic Alcoholic Intoxication," New York, 1868, p. 10.

In another instance I performed the converse experiment of exsecting the sciatic nerves, leaving the other tissues of the extremities intact. I then, as before, inserted both legs into absolute alcohol. No shock ensued, and the animal was not apparently affected by the alcohol till twenty-two minutes had elapsed.

Absorption of alcohol from the stomach is sometimes greatly delayed, and yet many of the effects of the substance are observed. Most of us have seen an intoxicated man relieved immediately by the full action of an emetic. Of course the emetic in such a case can only remove the non-absorbed alcohol still remaining in the stomach, and yet the symptoms of inebriation disappear on its ejection. It can only have acted through the nervous system, without the intermediation of the blood.

Observations and experiments such as these are very striking and important. They tend to show that the action of alcohol is exerted upon the nervous system in a twofold manner, and they are evidence of the remarkable affinity which the substance in question has for the nerve-tissue.

Post-mortem examinations of persons who have died directly from the effects of alcohol, or who were during life habitual drunkards, also show how powerfully the nerve-centres are influenced by this agent. In extreme cases it has not infrequently happened that the brain, on being exposed, has evolved a strong odor of alcohol. It is true that the experiments of Dr. Hutson Ford[1] appear to show that alcohol is a normal constituent of the blood; but it is very certain that the quantity is altogether too small to give the characteristic odor of this substance, although the reaction with chromic acid, and the distillate being capable of ignition and burning like alcohol, are affirmative evidences of great significance. He did not, however, examine the brain for alcohol, and my own experiments on this point, with the brains of dogs and oxen, and of men not addicted to the use of alcoholic liquors, have given negative results. Aware, however, of the great affinity which the cerebral and other nerve-tissues have for alcohol, it seems to me that if this substance is normally present in the blood it ought to be found as well in the brain as in the lungs and liver, unless, as may have been the case, the alcohol discovered by Dr. Ford in these organs and in the blood was a post-mortem production.

With the view of still further elucidating this subject, I fed a rabbit largely every day with bread soaked in whiskey. In the course of that time the animal received nearly a pint of the liquor, but beyond being somewhat stupefied it did not appear to be seriously inconvenienced. At the end of ten days the animal was killed.

I then removed the brain, the spinal cord, and all the large nerves, and treated them separately with distilled water after cutting them into small pieces. They were then thrown upon a filter and strongly pressed.

---

[1] "Normal Presence of Alcohol in the Blood," *Journal of the Elliott Society of Natural History*, vol. i., Charleston, 1859.

The three separate portions of liquid extract were then distilled several times, and finally treated with quicklime and again distilled. The odor of the distillates was almost sufficient of itself to establish the presence of alcohol, but, when the vapor from each was passed through the solution of bichromate of potash in sulphuric acid the characteristic green color resulting from the action of alcohol was at once produced.

So far as I am aware, no previous experiments had established the existence of alcohol in the spinal cord and the nerves.

A portion of the blood of the same animal treated in like manner failed to exhibit evidence of the presence of alcohol. The experiments, therefore, showed that the nervous tissue had a greater affinity for this substance than the blood.[1]

Besides the morbid conditions which exist in the nervous system as the direct result of the ingestion of alcohol in large quantities, this substance is capable of causing other patho-anatomical states which have already been described in this treatise.

Treatment.—In the first place, in the treatment of chronic alcoholism, the physician should insist upon entire cessation from the use of alcoholic liquors. It usually happens that the bowels are deranged by constipation or diarrhœa. In either case a mild purgative will be found of service. I know of nothing better than the following: ℞. Aloes, ext. fel. bovis exsic., āā grs. xv; resinæ podophilli, grs. ij. M. ft. in pill no. v. Dose, one every alternate day.

For the special treatment of the condition the oxide of zinc in doses of two or three grains three times a day has been strongly recommended by Marcet, and is certainly possessed of great power in this direction. Under its use the symptoms soon begin to disappear, and the patient to resume his normal condition of mind and body. But in my experience it is far inferior to the bromides of potassium, sodium, calcium, or ammonium, which, when given in doses of from fifteen to thirty grains in solution three times a day, are exceedingly efficacious. Even they, however, are inferior to the bromide of zinc, which may be administered in the dose of two grains in solution in water or simple syrup three or four times a day—gradually increased, as rapidly as the stomach will permit, to two or three times that quantity.

In some cases, especially in those in which insomnia is a prominent feature, the zinc compound may be advantageously given with either of the other bromides mentioned.

---

[1] These experiments were performed before the New York Neurological Society, May 4, 1874, and are detailed at length in the *Psychological and Medico-Legal Journal* for July, 1874.

I am very sure that in digitalis we have an important adjunct to the treatment mentioned. It not only acts as a tonic to the heart, but it is the most active agent we possess as an eliminant of alcohol through the kidneys. I prefer the infusion in doses of a tablespoonful three or four times a day. The tincture may be given in doses of from fifteen to thirty drops, as often.

In acute alcoholism, or delirium tremens, the treatment depends very much upon the mode of origin of the disease.

In those cases which have resulted from the sudden cessation from the use of alcoholic liquors, opium with brandy or whiskey should be given. The main indication is to procure sleep as soon as possible, and I am aware of no means so effectual in cases of this kind as the hypodermic injection of large doses of morphia—one-fourth to half a grain—as often as may be required, combined with the internal administration of brandy or whiskey in moderate quantities.

When, however, the affection has come on during a debauch, nothing can be much worse than either of those substances. They add fuel to the flame. In such cases the bromides, in large doses, combined with digitalis, are the most effective remedies. A drachm of the bromide of potassium, for instance, may be given in solution in a tablespoonful of infusion of digitalis every hour or two, and it will generally happen that sleep will follow, with the cessation or mitigation of all the permanent symptoms.

The hydrate of chloral has been recommended in delirium tremens, but I have no personal experience of its use.

The monobromide of camphor has been used successfully in delirium by M. Seneffe, of Belgium, and by Dr. O'Hara, of this country. I have also recently employed it in one case—administering four grains in capsule every hour. After the eighth dose the patient slept four hours. The remedy was again given as before, and after six doses another period of sleep, this time of six hours' duration, was obtained. The further administration was not necessary.

With the medical treatment in either form of delirium tremens the strength should be supported with beef-tea, and, after convalescence, quinine, iron, and strychnia, will prove of service.

CHAPTER III.

BROMISM.

In view of the facts that the bromides of potassium, sodium, calcium, lithium, and ammonium, are necessarily administered in several diseases of the nervous system, notably in epilepsy, in large doses and for long periods, and that a peculiar condition is thereby induced, it is important that the resultant phenomena should be recognized.

In adults it is rarely the case that any decided symptoms of bromism are caused by doses of less than thirty grains daily, and not often that forty-five grains a day produce them in any great intensity. In children, however, and sometimes in weak individuals, smaller quantities will give rise to very well-marked phenomena.

Symptoms.—The first symptom to make its appearance in cases of bromism is drowsiness. The patient sleeps not only at night, but in the day, and often under circumstances in which sleep would appear to be almost out of the question. Feebleness of the arms and legs, especially of the latter, is generally the next sign. The gait becomes titubating, and falls are apt to occur, especially in children. The grasp of the hands is weak, and there appears to be an anæsthesia of what may be called the muscular sense, for articles held are dropped unless the sight be kept upon them.

Articulation is very early interfered with, so that the speech becomes thick and indistinct. Words are omitted and others are clipped of their final syllables, or are slurred over in a tangled mass of incomprehensible utterances.

The action of the heart is weakened, and at the same time rendered more frequent; the skin is cold and clammy, the countenance is pale, and the pupils, from being at first somewhat contracted, become widely dilated and somewhat insensible to light.

The tongue is reddened, thickly coated, dry, and sometimes sore. The breath has the odor of bromine or is otherwise offensive; the bowels are usually constipated, and the urine is ordinarily increased in quantity.

The skin, even in cases in which the other symptoms of bromism are not very evident, is the seat of numerous pustules, especially that covering the face, neck, back, and chest, and occasionally large boils or carbuncles make their appearance.

The fauces are often intensely congested, and aphthous patches appear on the mucous membrane of the buccal cavity. The respiration becomes hurried, cough is often induced, and bronchitis or congestion of the lungs may ensue.

The sensibility of the pharynx is markedly impaired, and its reflex excitability is almost if not entirely abolished. It requires a mental effort for the patient to swallow, and manual irritation of the fauces fails to excite nausea or efforts to vomit.

Finally, locomotion becomes impossible, the patient is in a state of continual stupor, incapable of making known his wants—in fact, having no wants—and unable to recognize those about him ; the urine and fæces are passed involuntarily, the lungs are engorged, the heart becomes still weaker, and if the administration of the bromide be not suspended death ensues.

In a paper which I published[1] several years ago, I called attention to this remarkable condition, and adduced several cases in illustration of the points then brought forward. They were noticed by Huette[2] many years ago, though not very perfectly. He was the first to observe the effects of bromide of potassium upon the generative function in the abolition it causes of sexual desire and power.

Before the extreme influence of the bromides is attained, a patient under their influence presents phenomena very similar to those exhibited by a drunken person. A case which formed one of the series given in the paper referred to is so apposite in the present connection that I cite it here :

A gentleman consulted me in January, 1867, for severe headache, with which he had suffered for many years. He informed me that he had once fallen from the rigging of a vessel, had struck his head, and was rendered insensible for several hours. Subsequently he had a sunstroke in Texas. I considered this a suitable case for the administration of the bromide of potassium, and accordingly prescribed for him a teaspoonful three times a day of a solution containing one ounce of the medicine to four ounces of water. He thus took about fifteen grains at a dose. The effects of this were so pleasant to him, and yet not altogether so strong as he desired, that he began to increase the dose. Being absent from the city for two or three weeks at that time, I did not witness the phenomena. I was informed, however, that he had exhibited symptoms of mental aberration. These wore off on the cessation of the medicine, and when I returned he was comparatively well.

His headaches, however, soon came back with all their original violence, and at his earnest solicitation, and under his promise not to exceed the prescribed dose, I again gave him the bromide. He very soon began to increase the quantity, and finally seemed to have lost all control of his appetite for it. At this time I ascertained that he was in the habit of having his four-ounce vial containing an ounce of the bromide filled every day. The first obvious effect was an unsteadiness of gait. So great was this that he was frequently taken for a drunken man, and on one occasion was arrested by the police, confined in a cell all night, and fined the next morning, notwithstanding my statement of the facts to the police superintendent. On another occasion I met him in the street, as I was going to visit him. He was now decidedly insane; had delusions that lewd women had got into his mother's house; that he was pursued by the police; that his life was threatened by members of the family; that he had thousands of dollars of gold sewed up in his clothing, etc. When I met him his appearance and manner were very similar to those of a drunken man, except that his face was exceedingly pale. This gentleman was a total-abstinence man as regarded intoxicating liquors of all kinds. His manner was excited and rambling, and his hands were constantly busy either in fumbling in his pockets, tying his shoes, picking threads from his clothing, or in reaching for the gold which he believed was concealed in the lining of his coat. His character had also undergone a radical change. From having been very frank and brave, he had become excessively timid and suspicious of every trifling circumstance.

---

[1] "On some of the Effects of the Bromide of Potassium when administered in Large Doses," *Quarterly Journal of Psychological Medicine*, vol. iii., 1869, p. 46.

[2] *Gazette Médicale*, June, 1850.

Up to this period I was not quite sure that he was suffering from the effects of bromide of potassium. His symptoms were in many respects so much like those of an ordinary attack of acute mania, and his antecedents were of such a character as to predispose him to an accession of the kind, that I had reasons for my doubts. Nevertheless, I endeavored to stop his use of the bromide. This was a difficult task, for, notwithstanding all efforts, he continued to get hold of it. At last it was ascertained that he had secreted large quantities of it in various out-of-the-way places about the house.

His mental derangement had now become so prominent and constant that his friends became alarmed for his own and their safety. He had several times attempted to throw himself from the window, and had battered down a door with an axe in order to escape from some imaginary danger. Under these circumstances I recommended his committal to a lunatic asylum, and he was accordingly removed to Sanford Hall, at Flushing. Here his symptoms gradually disappeared, and in a month he returned to his home well. He has continued so to this day, with the exception that his headaches, which had disappeared while he was under the influence of the bromide, became as severe as at first, and still continue.

This was certainly an extreme case, but others fully as well marked have come under my notice.

The effects due to the continued administration of the bromide of potassium have not been more clearly, fully, and at the same time succinctly stated than by Dr. E. H. Clarke.[1] He says:

"The principal phenomena following the continued dose are: acne;

---

[1] "The Physiological and Therapeutical Action of the Bromide of Potassium and Bromide of Ammonium," by Edward H. Clarke, M. D., and Robert Amory, M. D., Boston, 872, p. 36.

salivation and salt taste in the mouth; irritation of the fauces, generally with œdema and redness, and sometimes with paleness of those parts; moderate anæsthesia of the pharynx; laryngo-bronchial weakness, sometimes with cough and sometimes with a changed or whispered voice, rarely with aphonia; a fetid or bromized breath; occasional stammering; increase of renal secretion; diminution of mucous secretion generally; slight constipation, and, in a few rare cases, diarrhœa; sense of mental and physical languor or weakness; sometimes temporary impairment of the memory, general aspect of hebetude or indifference; more or less somnolence; repression, and occasionally temporary abolition of sexual desire and power; impaired locomotion, which, when the dose is excessive, resembles the gait of locomotor ataxia; diminished nervous sensibility in general, and especially diminution of reflex sensibility; and, finally, an increase of destructive without a corresponding increase of constructive metamorphosis, and consequent emaciation."

When administered in larger quantities than are just sufficient to produce the foregoing symptoms, the phenomena, as detailed by Dr. Clarke, are:

"The fetid breath becomes nauseous; œdema supervenes on congestion of the uvula and fauces; the whispering voice sinks into aphonia; sexual weakness degenerates into impotence; muscular weakness becomes complete paralysis; reflex, general, and special sensations disappear; the ears do not hear, or the eyes see, or the tongue taste; the expression of hebetude becomes first that of imbecility, and then that of idiocy; hallucinations of sight and sound, with or without mania, precede general cerebral indifference, apathy, and paralysis; the respiration, without the stertor of opium or alcohol, is easy but slow; the temperature of the body is lowered; as the bromism becomes more profound, the patient lies quietly in his bed, unable to move, or to feel, or swallow or speak, with dilated and uncontractile pupils, and scarcely any change of the color of the skin or face; the extremities grow gradually colder and colder; the action of the heart becomes feeble and slower, till it ceases altogether."

Dr. Clarke reports one death, in which this result was probably due to bromism. Three have come under my observation, in which bromism was probably instrumental in hastening a fatal termination. In one of these the patient, a young lady, was the subject of epilepsy. She resided out of the city, and I prescribed the bromide of potassium in doses of fifteen grains three times a day. While taking it, and fully under its influence, she contracted pneumonia; but, without my knowledge, the medicine was continued, and she died.

The second case was that of a lady forty years of age, also subject to epilepsy, for whom I prescribed the bromide of sodium in doses of fifteen grains three times a day. The bromic cachexia soon became strongly marked, but, as I saw her every day, I did not think it advisable to reduce the doses. She went out every day, and on one occasion crossed the North River ferry to meet some friends. She caught a severe cold, pneumonia supervened, and, though the administration of the medicine was at once stopped, she died in the second stage of the disease.

In both these cases the bromide probably was indirectly the cause of death by the asthenia which it produced.

In the third case the patient, a lady from the South, also an epileptic, visited New York to consult me relative to her disease. I prescribed for her as in the last-named case, and, after remaining a fortnight in the city, she returned home with no great degree of bromism. But, after her departure, the toxic influence became more strongly marked, and, before I could be written to and my answer obtained, the medicine being continued all the time, death occurred. In this instance the result was doubtless entirely due to bromism.

Causes.—For the production of bromism, more or less prolonged administration of a bromide—the continued dose of Dr. Clarke—is necessary. In my experience the potassium and sodium salts equally cause it; the lithium, calcium, and ammonium compounds, less readily; the bromide of zinc not at all; but this result may be due to the fact that this preparation is not administered in as large doses as the others. Great differences exist among individuals in regard to the capacity to be brought under the full influence of a bromide; but I know of no signs by which these differences can be previously ascertained, except those of age and sex; children and women being more readily affected as a rule.

The administration of a bromide in a largely diluted form facilitates the action of the drug on the system, and consequently leads more readily to the promotion of bromism. This is probably due to the fact of its greater endosmotic power, and consequent more rapid absorption into the blood.

The **Diagnosis** of bromism scarcely calls for remark. The **Prognosis** is almost invariably favorable if the administration of the drug be stopped when the phenomena become profound and there are no serious superadded affections present.

Of the **Morbid Anatomy** nothing is known, and the **Pathology** is, therefore, based entirely on what has been ascertained relative to the physiological and therapeutic action of the bromic compounds. Chief among these are the facts that it diminishes the amount of blood circulating in the cerebral blood-vessels, and that it lessens the irritability of the whole cerebro-spinal and sympathetic nervous systems. These effects were set forth in a paper[1] published more than ten years ago, and have been generally confirmed by subsequent observers, and by myself in various memoirs.[1] Relative to the influence which the bromides exert in diminishing the quantity of blood in the brain, the fact admits of actual demonstration by means of inspection through the trephined skulls of animals and by the use of the cephalohæmometer described in the introduction to this work.

---

[1] "On Sleep and Insomnia," *New York Medical Journal*, 1865.

Many of the most striking phenomena of bromism are the result of the cerebral anæmia which the bromides produce. The paleness of the countenance, the dilatation of the pupils, the mental and physical weakness, the somnolence, the cardiac debility, all result from the intracranial condition.

Among the secondary effects are those cited by Bartholow :[2] the retardation of the process of destructive metamorphosis, the diminution of the sexual desire and power, and gastric derangement.

M. Laborde[3] has performed a number of experiments on man and other animals with the bromide of potassium. Four or five minutes after the administration of from three to six grains to frogs, a slight general excitement, with moderate tetanic movements, was produced. Weakness followed, and then there was a condition of flaccidity, during which reflex action was entirely abolished. The heart was but slightly affected, and continued to contract for several hours after this loss of reflex power. Laborde concludes, therefore, that bromide of potassium has no special action on the heart, muscles, encephalon, or nerves, but that it mainly and primarily injures the spinal cord.

These views are doubtless true as regards the frog, in which animal the spinal cord is mainly the seat of the mind, and therefore any cerebral influence must be very slightly manifested ; but they certainly are not correct so far as man and the superior animals are concerned.

Other observers have written relative to the physiological effects of the bromides, among whom MM. Damourette and Pelvet[4] may be mentioned.

In the work of Drs. Clarke and Amory, to which reference has already been made, Dr. Amory enunciates, among other propositions, the following :

"The loss of reflex action is due to the diminution of blood in the periphery of the nerves and also of the central nervous system, this last occurring after the first.

"The action of bromide of potassium on the nervous system may be explained by its action on the capillary, arterial, or central circulation."

These propositions are supported by various experiments, and appear to be well established.

---

[1] "On some of the Effects of the Bromide of Potassium," etc., *Quarterly Journal of Psychological Medicine*, January, 1869.

[2] *Cincinnati Lancet and Observer*, 1865.

[3] *Comptes Rendus*, July 8, 1868.

[4] *Bulletin générale de thérapeutique*, 1867, pp. 241, 289.

The recent work of Voisin[1] adds nothing to our previous knowledge of the subject.

Treatment.—There is no special treatment for bromism beyond that which consists in suspending at once the administration of the medicine, facilitating its elimination from the system, and sustaining the strength. Dr. Clarke[2] has shown that the fæces do not contain an appreciable quantity of the bromide of potassium, even when it is being taken in large quantity. He found that it is mainly eliminated by the kidneys and by the skin. It is difficult to avoid the opinion, in view of the odor of the breath of persons taking a bromide, that bromine is eliminated with the expired air, but Dr. Clarke's experiments appear to establish the negative. The indication, therefore, is to administer diuretics and diaphoretics. Nothing is better for the first than digitalis, which not only acts upon the kidneys, but is also a tonic to the heart, and for the latter than warm drinks, such as infusion of flaxseed, lemonade, etc., which are also more or less diuretic.

The strength of the patient should be sustained with brandy or wine, quinine, beef-tea, etc.

CHAPTER IV.

HYDRARGYSM.

Symptoms.—The consequences to the nervous system, from the slow absorption of mercury into the organism, have been known for many years. The principal phenomenon witnessed is tremor, but there are other symptoms which serve for the recognition of the nature of the disorder.

Thus the gums are swollen and tender, the breath fetid, the teeth become loose, especially those of the lower jaw, and there is a metallic taste in the mouth. The lining membrane of the mouth and throat becomes inflamed, and ulcerations very generally occur in the fauces. The quantity of saliva is greatly increased.

These symptoms exist mainly in the first stage of hydrargysm, and constitute what is generally called salivation. But, if the mercury continues to be taken into the system, another series of phenomena appears. Or, if the absorption has been extremely slow, the foregoing may be in great part, or entirely, absent.

The symptoms referred to are paleness or lividity of the countenance, the frequent occurrence of nasal hæmorrhages, and marked mental weakness. The physical strength gradually becomes less, and tremor makes its appearance, mostly confined, in the early stages at least, to the superior extremities and the head. Finally, the lower limbs are affected, and, in addition, are generally the seat of œdema. Pains in the bones, and caries, and necrosis, especially of the maxillary bones, may occur together with ulcerations of the soft parts.

---

[1] "De l'emploi de bromure de potassium dans les maladies nerveuses," Paris, 1875.

[2] *Op. cit.*, p. 139.

The mental symptoms are generally strongly marked. There are hallucinations and delusions, accompanied sometimes with a high degree of maniacal excitement. Epileptiform convulsions may occur, as may also paralysis of various parts of the body, and finally, unless relief be afforded, death ensues.

Causes.—Mercury may be taken into the system and be the cause of slow poisoning, through the skin, the stomach and intestines, and the lungs. Fire-gilders, looking-glass manufacturers, barometer-makers, the workers in quicksilver-mines, bronzers, the makers of artificial flowers, and photographers, exposed as they are to the vapor, the fine powder, or a solution containing mercury, are therefore liable to its toxic influence. Hydrargysm has also been known to be induced by the long-continued administration of preparations of mercury in medical practice, and even from the filling of a tooth with an amalgam.

I have known of a case of mercurial tremor, produced in a young lady by the use of a solution of corrosive sublimate as a cosmetic to remove pimples from the face.

The **Diagnosis** of hydrargysm is in general much elucidated by the clinical history of the case and the knowledge that the patient has been exposed to mercurial emanations. In addition, the tremor, the fetid breath, loosening of the teeth, caries of the bones, and the muscular weakness, are diagnostic signs of value, while the absence of the blue line on the gums—although it is stated that such a line is sometimes present—and the fact that the extensors are not especially the seat of paralysis will suffice for the discrimination of hydrargysm from plumbism.

But the diagnosis is rendered quite certain by the administration of the iodide of potassium, which, as Melsens has shown, separates mercury from its combinations with the tissues of the body, forming with it a new compound—the iodide of mercury—which is eliminated with the urine. All that is necessary, therefore, is to give the iodide of potassium in large doses to a patient suspected to be suffering from hydrargysm, to put a few drops of the urine, excreted during the second day, on a bright copper plate, and then add a drop of hydrochloric acid. A bright metallic stain will be found on the plate if mercury be present. The iodide of mercury is decomposed and the metal is precipitated as stated.

The **Prognosis** is generally favorable if the patient can be removed from further contamination with mercury and be subjected to proper treatment.

Morbid Anatomy and Pathology.—There are no data by which we can form an opinion relative to the anatomical changes induced in the nerve-tissues by the action of mercury. It is probable, as M. Sée asserts, that this substance, like lead, forms an albuminate of mercury both in the blood and the solid parts of the body. Beyond this fact we have nothing except the gross alterations found in the stomach, the kidneys, and other organs, when mercury has been taken into the system in large enough quantities to cause death. It is perhaps scarcely necessary to say that the accounts which have been given of metallic mercury being found in globules in the brain and other organs are not correct.

A curious circumstance, which has sometimes been observed, is the occurrence of salivation in the cases of persons who have taken mercury, but who have not exhibited any indications of hydrargysm previous to the administration of iodide of potassium. It appears that the mercury set free from its albuminate compounds is enabled, while traversing the system in its exit through the kidneys, to exert a toxic power. I have never witnessed cases of the kind, and they must be rare. It will be recollected that a like action is claimed for lead.

Treatment.—The special means of treatment consists in the free administration of the iodide of potassium in accordance with the discovery of M. Melsens, already alluded to. Under the action of this remedy the symptoms of hydrargysm speedily disappear, and the patient regains his normal or almost his normal condition. The worst case of the affection that has come within my experience, was that of a looking-glass maker of this city, in whom the tremor and other evidences of cachexia were exceedingly striking. He was unable to write, from paralysis, and barely able to shuffle about his room. I gave him at once thirty grains of the iodide a day, in divided doses, and in the course of a week doubled the quantity. He immediately began to mend, and was well in less than five weeks.

Of course, while under treatment the patient must not be subjected to continual poisoning from mercury.

Tonics—iron, quinine, and strychnia—are useful adjuncts.

CHAPTER V.

ARSENICISM.

THE **Symptoms** indicative of slow poisoning by arsenic are generally quite characteristic. There are vomiting, a sensation of heat in the throat and stomach, colicky pains, weakness of the limbs, tremor, vertigo, hæmorrhage from the nostrils, puffiness of the face, especially about the eyes, attacks of syncope or of epileptiform convulsions, pains in the joints and contractions of the fingers and toes, numbness sometimes amounting to complete anæsthesia, and paraplegia.

In addition there are sometimes ophthalmia and various papular and vesicular eruptions on the skin.

Death is the almost inevitable consequence if the exposure to toxication continues, or speedy relief be not afforded by medical treatment.

The **Causes** of arsenical cachexia are, like those of lead and mercury, to be found generally with those whose occupation requires exposure to contact, through the lungs, skin, or alimentary canal, with arsenical preparations. It is thus met with in furriers, who use arsenious acid as a preservative; in taxidermists, who employ it for a like purpose; in naturalists, who sprinkle it over their zoölogical specimens, and who in handling them absorb the powder through the lungs; in the manufacturers of paper-hangings; in dressmakers, who are obliged to handle green tarlatan ; in makers of artificial flowers, and in the workers in chemical manufactories, where arsenical preparations are made; and in those who labor in arsenic-mines.

It has also occurred in persons occupying rooms hung with the brilliant green-velvet paper into the manufacture of which arsenic enters in large quantities.

Notwithstanding the general susceptibility of mankind to the deleterious influence of arsenic, it appears that the system may become so habituated to its use as actually to thrive under it. This is the case with the arsenic-eaters of Styria, who take habitually from two to four or five grains daily, and who, nevertheless, are extremely healthy, and even rugged-looking people.

The **Diagnosis** is not a matter of difficulty, especially if the clinical history be inquired into, and the **Prognosis**, except in extreme cases, is not unfavorable after the patient is removed from further contact with arsenic.

The **Morbid Anatomy and Pathology,** so far as the nervous system is concerned, are not known, and except in acute cases of arsenical poisoning, with which, however, we are not now concerned, there are no definite lesions discoverable in other parts of the body.

The **Treatment** consists in removing the patient from further exposure, and subjecting him to the most favorable hygienic influences, the strength being maintained by tonics. If there are contractions of any of the limbs, passive motion, frictions, and electricity, are indicated.

INDEX.

PAGE

Abdominal sympathetic, pathology of... 878
Abscess, chronic cerebral ... 275
Active cerebral congestion ... 33
  varieties of ... 33
  symptoms ... 34
  causes ... 56
  diagnosis ... 59
  prognosis ... 61
  morbid anatomy ... 62
  pathology ... 63
  treatment ... 65
Acute alcoholism ... 899
  symptoms ... 899
Acute cerebral meningitis ... 221
  symptoms ... 221
  causes ... 225
  diagnosis ... 226
  prognosis ... 226
  morbid anatomy ... 227
  pathology ... 227
  treatment ... 229
Acute general myelitis ... 455
  symptoms ... 455
Acute myelitis ... 455
Acute neuritis ... 825
  symptoms ... 825
  causes ... 826
  diagnosis ... 826
  prognosis ... 826
  morbid anatomy and pathology ... 826
  treatment ... 827
Acute partial myelitis ... 457
  symptoms ... 457
  causes ... 459
  diagnosis ... 460
  prognosis ... 461
  morbid anatomy and pathology ... 461
  treatment ... 462
Acute spinal meningitis ... 439

PAGE

Acute spinal meningitis, symptoms ... 439
Affections, hysteroid ... 765
Alcoholism ... 894
  symptoms ... 894
  causes ... 900
  diagnosis ... 902
  prognosis ... 902
  morbid anatomy and pathology ... 902
  treatment ... 911
Alcoholism, acute ... 899
Alcoholism, chronic ... 894
Amyotrophic lateral spinal sclerosis ... 592
  symptoms ... 593
  causes ... 596
  diagnosis ... 596
  prognosis ... 597
  morbid anatomy and pathology ... 597
  treatment ... 603
Anæmia, cerebral ... 71
Anæmia, partial cerebral ... 131
Anæmia, spinal ... 399
Anæmia of antero-lateral columns of cord 423
  symptoms ... 424
  diagnosis ... 425
  prognosis ... 425
  morbid anatomy and pathology ... 426
  treatment ... 430
Anæsthesia, lead ... 838
Anæsthesia, neural ... 845
Anæsthesia of cutaneous nerves ... 845
  symptoms ... 845
  causes ... 847
  diagnosis ... 847
  prognosis ... 847
  morbid anatomy and pathology ... 848
  treatment ... 848
Anæsthesia of fifth pair ... 848
  symptoms ... 848
  causes ... 849

PAGE
Anaesthesia of fifth pair, diagnosis...... 849
  prognosis.......................... 849
  morbid anatomy and pathology..... 849
  treatment.......................... 849
Anapeiratic paralysis.................. 807
  symptoms.......................... 808
  causes............................ 810
  diagnosis.......................... 811
  prognosis.......................... 811
  morbid anatomy and pathology..... 811
  treatment.......................... 811
Anterior and posterior tracts of gray matter, inflammation of.............. 573
Anterior columns, inflammation of...... 586
Anterior tract of gray matter, inflammation of.......................... 463
Antero-lateral columns of the cord, anæmia of.......................... 423
Aphasia................................ 181
Arsenicism............................ 921
  symptoms.......................... 921
  causes............................ 922
  diagnosis.......................... 922
  prognosis.......................... 922
  morbid anatomy and pathology..... 922
  treatment.......................... 922
Athetosis.............................. 325
Atrophy, neural....................... 828
Atrophy of the cerebellum.............. 378

Basilar meningitis, chronic............. 241
Brain, syphilis of............. 342
Brain, tumors of....................... 308
Bromism.............................. 913
  symptoms.......................... 913
  causes............................ 917
  diagnosis.......................... 917
  prognosis.......................... 917
  morbid anatomy.................... 917
  treatment.......................... 919

Catalepsy.............................. 765
  symptoms.......................... 766
  causes............................ 771
  diagnosis.......................... 771
  prognosis.......................... 772
  morbid anatomy and pathology..... 772
  treatment.......................... 773
Cerebellar diseases..................... 377
    hæmorrhages................ 372
    symptomatology of........... 372
Cerebellum, atrophy of 377
Cerebellum, tumors of.................. 375
Cerebral abscess....................... 275
Cerebral anæmia....................... 71
  symptoms.......................... 71
  causes 74
  diagnosis.......................... 76

PAGE
Cerebral anæmia, prognosis............. 76
  morbid anatomy.................... 77
  pathology.......................... 77
  treatment.......................... 78
Cerebral anæmia, partial............... 131
Cerebral arteries, embolism of.......... 141
Cerebral arteries, thrombosis of......... 131
Cerebral blood-vessels, obliteration of... 131
Cerebral capillaries, embolism and thrombosis of.......................... 153
Cerebral and cerebellar peduncles, lesions of.......................... 371
Cerebral congestion..................... 33
Cerebral congestion, active............. 33
Cerebral congestion, passive............ 40
Cerebral hæmorrhage.................. 81
  symptoms.......................... 82
  causes............................ 93
  diagnosis.......................... 96
  prognosis.......................... 100
  morbid anatomy.................... 101
  pathology.......................... 105
  treatment.......................... 108
Cerebral meningeal hæmorrhage........ 123
  symptoms.......................... 124
  causes............................ 125
  diagnosis.......................... 126
  prognosis.......................... 126
  morbid anatomy and pathology..... 127
  treatment.......................... 129
Cerebral meningitis, acute.............. 220
Cerebral meningitis, chronic............ 230
Cerebral softening...................... 160
  symptoms.......................... 160
  causes............................ 165
  diagnosis.......................... 168
  prognosis.......................... 169
  morbid anatomy.................... 169
  pathology.......................... 171
  treatment.......................... 175
Cerebral sclerosis, diffused............ 280
Cerebral sclerosis, multiple............ 291
Cerebral syphilis...................... 342
Cerebria.............................. 278
Cerebritis............................. 263
Cerebro-spinal diseases................. 664
Cervical hypertrophic pachymeningitis.. 429
Cervical sympathetic, neuroses of....... 868
Cervical sympathetic, pathology of...... 864
Cervico-brachial neuralgia.............. 853
Cervico-occipital neuralgia............. 852
Chorea................ 732
  symptoms.......................... 732
  causes 738
  diagnosis.......................... 740
  prognosis.......................... 740
  morbid anatomy and pathology..... 741
  treatment.......................... 743

PAGE

- Chronic alcoholism 894
  - symptoms 894
- Chronic basilar meningitis 241
  - symptoms 242
  - causes 249
  - diagnosis 256
  - prognosis 256
  - morbid anatomy 251
  - pathology 253
  - treatment 257
- Chronic cerebral abscess 259
- Chronic cerebral meningitis 231
- Chronic neuritis 828
  - symptoms 828
  - causes 829
  - diagnosis 830
  - prognosis 830
  - morbid anatomy and pathology 830
  - treatment 830
- Chronic spinal meningitis 440
  - symptoms 440
  - causes 441
  - diagnosis 442
  - prognosis 442
  - morbid anatomy and pathology 442
  - treatment 449
- Chronic verticalar meningitis 231
  - symptoms 231
  - causes 236
  - diagnosis 237
  - prognosis 237
  - morbid anatomy and pathology 238
  - treatment 240
- Colic, lead 887
- Columns, anterior, inflammation of 586
- Columns of Goll, sclerosis of 636
- Columns, lateral and gray matter, inflammation of 592
- Columns, lateral, inflammation of 587
- Columns of Türck 586
- Congestion, cerebral 83
- Congestion, neural 823
- Congestion, spinal 391
- Convulsive tremor 720
  - history and symptoms 720
  - causes 728
  - diagnosis 729
  - prognosis 729
  - morbid anatomy and pathology 729
  - treatment 731
- Cortical paralysis 351
- Crural neuralgia 854
- Cutaneous nerves, anæsthesia of 845

- Diffused cerebral sclerosis 281
  - symptoms 281
  - causes 289
  - diagnosis 289

PAGE

- Diffused cerebral sclerosis, prognosis 290
  - morbid anatomy 290
  - pathology 291
  - treatment 291
- Diseases of peripheral nervous system 822
- Diseases of the sympathetic nervous system 864
- Disseminated inflammation of the spinal cord 638
  - symptoms 638
  - causes 640
  - diagnosis 641
  - prognosis 641
  - morbid anatomy and pathology 641
  - treatment 644
- Dorso-intercostal neuralgia 853
- Dura mater, pachymeningitis and hæmatoma of 130

- Ecstasy 775
  - symptoms 775
  - causes 785
  - treatment 785
- Embolism of cerebral arteries 142
  - symptoms 142
  - causes 145
  - diagnosis 145
  - prognosis 147
  - morbid anatomy and pathology 147
  - treatment 149
- Embolism of cerebral capillaries 154
- Embolism, fat 157
- Embolism, pigment 155
- Encephalitis, suppurative 269
- Epilepsy 685
  - symptoms 685
  - causes 698
  - diagnosis 700
  - prognosis 701
  - morbid anatomy 701
  - pathology 703
  - treatment 714
- Exophthalmic goitre 812
  - symptoms 812
  - causes 817
  - diagnosis 818
  - prognosis 818
  - morbid anatomy and pathology 818
  - treatment 820

- Facial atrophy, progressive 558
- Facial paralysis 832
  - symptoms 832
  - causes 836
  - diagnosis 836
  - prognosis 836
  - morbid anatomy and pathology 837
  - treatment 837

PAGE

- Facial spasm.......................... 842
  - symptoms.......................... 842
  - causes.......................... 842
  - diagnosis.......................... 843
  - prognosis.......................... 843
  - morbid anatomy and pathology..... 843
  - treatment.......................... 843
- Fat embolism.......................... 157
- Fifth pair, anæsthesia of.............. 848
- Fifth pair of nerves, neuralgia of........ 850

- General acute myelitis................ 455
- Glosso-labio-laryngeal paralysis......... 517
  - symptoms.......................... 518
  - causes.......................... 524
  - diagnosis.......................... 524
  - prognosis.......................... 525
  - morbid anatomy and pathology..... 526
  - treatment.......................... 532
- Goll, sclerosis of columns of............ 636

- Hæmatoma of the dura mater........... 130
  - symptoms.......................... 130
  - causes.......................... 130
  - diagnosis.......................... 131
  - prognosis.......................... 131
  - morbid anatomy and pathology..... 131
  - treatment.......................... 132
- Hæmorrhage, cerebellar................ 377
- Hæmorrhage, cerebral.................. 81
- Hæmorrhage, cerebral meningeal........ 124
- Hæmorrhage, meningeal................ 432
- Hæmorrhage, spinal.................... 432
- Hemicrania.......................... 868
- Hemisphere, paralyses from central lesions of.......................... 362
- Hydrargysm.......................... 919
  - symptoms.......................... 919
  - causes.......................... 920
  - diagnosis.......................... 920
  - prognosis.......................... 920
  - morbid anatomy and pathology..... 920
  - treatment.......................... 921
- Hydrophobia.......................... 664
  - symptoms.......................... 664
  - causes.......................... 671
  - diagnosis.......................... 673
  - prognosis.......................... 675
  - morbid anatomy.................. 676
  - pathology.......................... 681
  - treatment.......................... 683
- Hyperæsthesia, lead.................... 888
- Hyperæsthesia, neural.................. 849
- Hysteria.......................... 750
  - symptoms.......................... 750
  - causes.......................... 759
  - diagnosis.......................... 760
  - prognosis.......................... 760

PAGE

- Hysteria, morbid anatomy and pathology 761
  - treatment.......................... 762
- Hystero-epilepsy...................... 786
  - symptoms.......................... 786
  - causes.......................... 792
  - diagnosis.......................... 792
  - prognosis.......................... 792
  - morbid anatomy and pathology..... 792
  - treatment.......................... 792
- Hysteroid affections.................... 765

- Infantile spinal paralysis............... 465
  - symptoms.......................... 465
  - causes.......................... 468
  - diagnosis.......................... 468
  - prognosis.......................... 469
  - morbid anatomy.................. 469
  - pathology.......................... 478
  - treatment.......................... 480
- Inflammation of anterior columns....... 586
- Inflammation of anterior tract of gray matter.......................... 463
- Inflammation of anterior and posterior tracts of gray matter............. 573
- Inflammation of columns of Goll........ 636
- Inflammation of lateral columns........ 587
- Inflammation of motor cells............ 517
- Inflammation of motor and trophic cells. 464
- Inflammation of posterior tract of gray matter.......................... 571
- Inflammation of the spinal cord........ 455
- Inflammation of trophic cells........... 553
- Intra-spinal hæmorrhage and pachymeningitis.......................... 443

- Lateral columns and anterior gray matter, inflammation of.............. 592
- Lateral columns, inflammation of....... 587
- Lead anæsthesia...................... 888
- Lead-colic.......................... 887
  - symptoms.......................... 887
- Lead encephalopathy.................. 884
- Lead hyperæsthesia.................... 888
  - causes.......................... 889
  - diagnosis.......................... 889
  - prognosis.......................... 891
  - morbid anatomy and pathology..... 891
  - treatment.......................... 893
- Lead-paralysis........................ 886
- Lesions of the cerebral and cerebellar peduncles.......................... 371
- Lesions of optic tracts.................. 370
- Lesions of tubercula quadrigemina...... 367
- Locomotor ataxia...................... 603
- Lumbo-abdominal neuralgia............ 852

- Meningitis, acute cerebral.............. 221
  - chronic basilar.................... 241

PAGE

- Meningitis, chronic cerebral 231
  - chronic verticalar 231
  - rheumatic 224
  - senile 225
  - spinal 439
  - tubercular cerebral 261
- Migraine 868
- Motor cells, inflammation of 517
- Motor and trophic cells, inflammation of. 464
- Multiple cerebral sclerosis 292
  - symptoms 293
  - causes 299
  - diagnosis 300
  - morbid anatomy 301
  - pathology 302
  - treatment 305
- Multiple cerebro-spinal sclerosis 793
  - symptoms 793
  - causes 802
  - diagnosis 803
  - prognosis 803
  - morbid anatomy and pathology 803
  - treatment 803
- Multiple spinal sclerosis 638
- Muscular atrophy, progressive 534
- Myelitis, acute general 455
  - acute 455
- Myxœdema 334
  - symptoms 335
  - causes 340
  - diagnosis 341
  - prognosis 341
  - morbid anatomy and pathology 341
  - treatment 342

- Nerves, tumors of 831
- Nervous system, toxic diseases of 884
- Neural anæsthesia 845
  - atrophy 828
  - congestion 823
  - hyperæsthesia 849
  - sclerosis 828
  - spasm 842
  - paralysis 832
- Neuralgia 849
  - cervico-brachial 853
  - symptoms 853
  - causes 853
  - diagnosis 853
  - prognosis 853
- Neuralgia, cervico-occipital 852
  - symptoms 852
  - causes 852
  - diagnosis 852
  - prognosis 852
- Neuralgia, crural 854
  - dorso-intercostal 853
- Neuralgia of fifth pair of nerves 850
- Neuralgia of fifth pair of nerves, symptoms 850
  - causes 851
  - diagnosis 852
  - prognosis 852
  - lumbo-abdominal 852
  - sciatic 854
  - treatment of 855
- Neuritis, acute 825
  - chronic 828
- Non-inflammatory softening of the spinal cord 649
  - symptoms 649
  - causes 652
  - diagnosis 652
  - prognosis 652
  - morbid anatomy and pathology 653
  - treatment 653
- Nothnagel's symptomatology 380

- Obliteration of cerebral blood-vessels ... 132
- Optic tracts, lesions of 370
- Organic infantile paralysis 465

- Pachymeningitis 130
  - cervical 443
- Paralysis agitans 804
  - symptoms 805
  - causes 805
  - diagnosis 806
  - prognosis 806
  - morbid anatomy and pathology 806
  - treatment 807
- Paralysis, anapeiratic 807
  - symptoms 808
  - causes 810
  - diagnosis 810
  - prognosis 810
  - morbid anatomy and pathology 810
  - treatment 816
- Paralysis consecutive to central lesions of the hemispheres 362
- Paralysis, cortical 351
- Paralysis, facial 832
- Paralysis, glosso-labio-laryngeal 518
- Paralysis, neural 832
- Paralysis of radial nerve 841
- Paralysis of sixth nerve 841
- Paralysis of third nerve 839
- Paralysis, pseudo-hypertrophic spinal ... 504
- Paralysis, spinal, of adults 484
- Paralysis, lead 886
  - symptoms 886
- Partial cerebral anæmia 132
- Passive cerebral congestion 54
  - symptoms 54
  - causes 56
  - diagnosis 59

PAGE
Passive cerebral congestion, prognosis... 61
  morbid anatomy.................. 62
  pathology........................ 63
  treatment........................ 65
Pathology of abdominal sympathetic.... 878
Pathology of cervical sympathetic...... 864
Pathology of thoracic sympathetic...... 876
Peripheral nervous system, diseases of.. 822
Pigment, embolism.................. 155
Plumbism 884
  symptoms......................... 884
Posterior root-zones, sclerosis of........ 603
Posterior tract of gray matter, inflammation of.......................... 571
Primary symmetrical lateral sclerosis... 587
  symptoms......................... 587
  causes........................... 589
  diagnosis........................ 589
  prognosis........................ 590
  morbid anatomy and pathology..... 591
  treatment 591
Progressive facial atrophy.............. 558
  symptoms......................... 558
  causes 563
  diagnosis........................ 563
  prognosis........................ 563
  morbid anatomy and pathology..... 564
  treatment........................ 570
Progressive locomotor ataxia........... 603
  symptoms......................... 603
  causes 615
  diagnosis........................ 615
  prognosis 616
  morbid anatomy................... 616
  pathology........................ 621
  treatment........................ 632
Progressive muscular atrophy.......... 534
  symptoms......................... 534
  causes........................... 540
  diagnosis........................ 548
  prognosis........................ 549
  morbid anatomy and pathology..... 549
  treatment........................ 557
Pseudo-hypertrophic spinal paralysis... 504
  symptoms......................... 505
  causes 510
  diagnosis........................ 510
  prognosis 511
  morbid anatomy and pathology..... 511
  treatment........................ 516

Radial nerve, paralysis of.............. 841
Rheumatic meningitis.................. 224

Sciatica.............................. 854
  symptoms......................... 854
  causes 854
  diagnosis........................ 855

PAGE
Sciatica, prognosis..................... 855
  morbid anatomy and pathology..... 855
Sciatic neuralgia....................... 854
Sclerosis, amyotrophic, lateral spinal.... 592
Sclerosis, diffused cerebral.............. 281
Sclerosis, multiple cerebral............. 292
Sclerosis, multiple cerebro-spinal....... 793
Sclerosis, multiple spinal............... 638
Sclerosis, neural....................... 823
Sclerosis, primary symmetrical lateral... 587
Sclerosis of posterior root-zones........ 603
Secondary degeneration of spinal cord... 644
  symptoms......................... 645
  causes 647
  diagnosis 647
  prognosis 647
  morbid anatomy and pathology..... 647
  treatment........................ 648
Senile meningitis...................... 225
Sixth nerve, paralysis of............... 841
Softening, cerebral.................... 161
Softening, non-inflammatory, of spinal cord............................ 649
Spasm, facial.......................... 842
  neural........................... 842
Spastic spinal paralysis................ 587
Spinal anæmia......................... 399
Spinal congestion...................... 391
  symptoms......................... 391
  causes 393
  diagnosis........................ 395
  prognosis 395
  morbid anatomy................... 396
  pathology........................ 396
  treatment 397
Spinal cord, anæmia of posterior columns of............................. 399
  diseases of...................... 381
  disseminated inflammation of...... 585
  inflammation of.................. 455
  non-inflammatory softening of..... 649
  secondary degeneration of........ 644
  syphilis of...................... 661
  tumors of........................ 654
Spinal hæmorrhage..................... 432
  symptoms......................... 432
  causes 433
  diagnosis........................ 433
  prognosis 435
  morbid anatomy and pathology..... 437
  treatment 438
Spinal irritation....................... 409
  history.......................... 409
  symptoms......................... 410
  causes 413
  morbid anatomy and pathology..... 414
  diagnosis........................ 416
  prognosis 417

PAGE
- **Spinal** irritation, treatment 418
- **Spinal** meningitis 439
- **Spinal** paralysis, infantile 465
- **Spinal** paralysis of adults 484
  - symptoms 486
  - causes 494
  - diagnosis 495
  - prognosis 497
  - morbid anatomy and pathology 497
  - treatment 499
- **Suppurative** encephalitis 269
  - symptoms 269
  - causes 272
  - diagnosis 273
  - prognosis 273
  - morbid anatomy and pathology 274
  - treatment 278
- **Symmetrical** gangrene of the extremities 880
- **Sympathetic** nervous system, diseases of 864
- **Symptomatology** of cerebellar lesions 372
- **Symptomatology** of cerebral lesions 351
- **Syphilis**, cerebral 842
- **Syphilis** of the peripheral nervous system 862
- **Syphilis** of the spinal cord and its membranes 661

- **Tetanus** 573
  - symptoms 573
  - causes 576
  - diagnosis 577
  - prognosis 578
  - morbid anatomy and pathology 578
  - treatment 583
- **Third** nerve, paralysis of 839
  - symptoms 839
  - causes 840
  - diagnosis 840
  - prognosis 840
  - morbid anatomy and pathology 841
  - treatment 841
- **Thoracic** sympathetic, pathology of 876
- **Thrombosis** of cerebral arteries 130
  - symptoms 131
  - causes 134
  - diagnosis 135

PAGE
- **Thrombosis** of cerebral arteries, prognosis 135
  - morbid anatomy and pathology 136
  - treatment 140
  - cerebral veins and capillaries 154
  - cerebral capillaries 159
- **Thrombosis** of cerebral veins and sinuses 149
  - symptoms 149
  - causes 153
  - prognosis 153
  - morbid anatomy and pathology 154
- **Torticollis** 843
  - causes 844
  - diagnosis 844
  - prognosis 844
  - morbid anatomy and pathology 844
  - treatment 844
- **Toxic** diseases of the nervous system 884
- **Treatment** of neuralgia 855
- **Tremor**, convulsive 720
- **Trophic** cells, inflammation of 533
- **Tubercula** quadrigemina, lesions of 367
- **Tubercular** cerebral meningitis 261
  - symptoms 261
  - causes 265
  - diagnosis 266
  - prognosis 266
  - morbid anatomy and pathology 267
  - treatment 268
- **Tumors** of the brain 303
  - symptoms 308
  - causes 315
  - diagnosis 316
  - prognosis 318
  - treatment 323
- **Tumors** of cerebellum 375
- **Tumors** of nerves 831
- **Tumors** of spinal cord 654
  - symptoms 654
  - causes 659
  - diagnosis 659
  - prognosis 660
  - morbid anatomy and pathology 660
  - treatment 660
- **Türck**, columns of 586

- Veins and sinuses, cerebral, thrombosis of 149

THE END.

h

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

L341
H227
1881

Hammond, W. A. 725
A treatise on the diseases of the nervous system. 7th ed.

NAME	DATE DUE